THE
BRITISH JOURNAL OF SURGERY

VOL. XXVII APRIL, 1940 No. 108

In Memoriam

WILFRED TROTTER

(1872–1939)

WILFRED TROTTER died on November 25, 1939, at the age of 67. His death has deprived the world of one of the most contemplative minds that has ever been trained towards surgery. His penetrating intellect delighted in an impersonal activity of thought which had as raw material its own original observation of the workings of men's minds and of the sources of their conduct. The main stream of his life ran always towards the pursuit of truth, and his interest in surgery was the joy of a fine intellect in the practice of a worthy handicraft.

Trotter was reticent about the effect of early education on the moulding of his mind and gave the impression that he showed no special aptitudes at school. From his conversation it was clear that the flowering of his latent genius was initiated by his first contact with the scientific method when he entered University College, London, at the age of 18. The vividness of this experience was so present in his mind as afterwards to determine the conviction that the first introduction to medicine of the receptive student mind should be made by teachers who were themselves engaged in the practice of the scientific method.

In his university final examinations he obtained honours in medicine and the gold medal in surgery, and by the age of 27 he had completed his examinations for the higher degrees. Then followed two years as Surgical Registrar at University College Hospital, during which his surgical ideas began to crystallize. He had been House Surgeon to Arthur Barker, whose natural ability in surgical technique was enhanced by frequent contacts with Continental surgeons. Barker's receptivity to new ideas and lifelong search for a wider surgical knowledge, though sometimes marred by trivialities which excited derision in lesser minds, commanded Trotter's constant respect. Throughout his life he paid frequent tribute to Barker's singleness of purpose and greatness of surgical stature.

When Trotter's Registrarship came to an end there was no immediate prospect of a vacancy on the surgical staff of the Hospital and he returned to University College as a Demonstrator of Anatomy under George Thane. The three years that followed brought him a leisure which he filled in characteristic fashion by the

dual exercise of philosophic thought and of the scientific method. The fruit of this most fertile period of his life was published during the years 1907–9, his two classical papers on "The Herd Instinct", in the *Sociological Review*, and on "Experimental Studies in the Innervation of the Skin", in the *Journal of Physiology*. The latter was written in collaboration with Morriston Davies and embodied their conclusions from the section of their own cutaneous nerves.

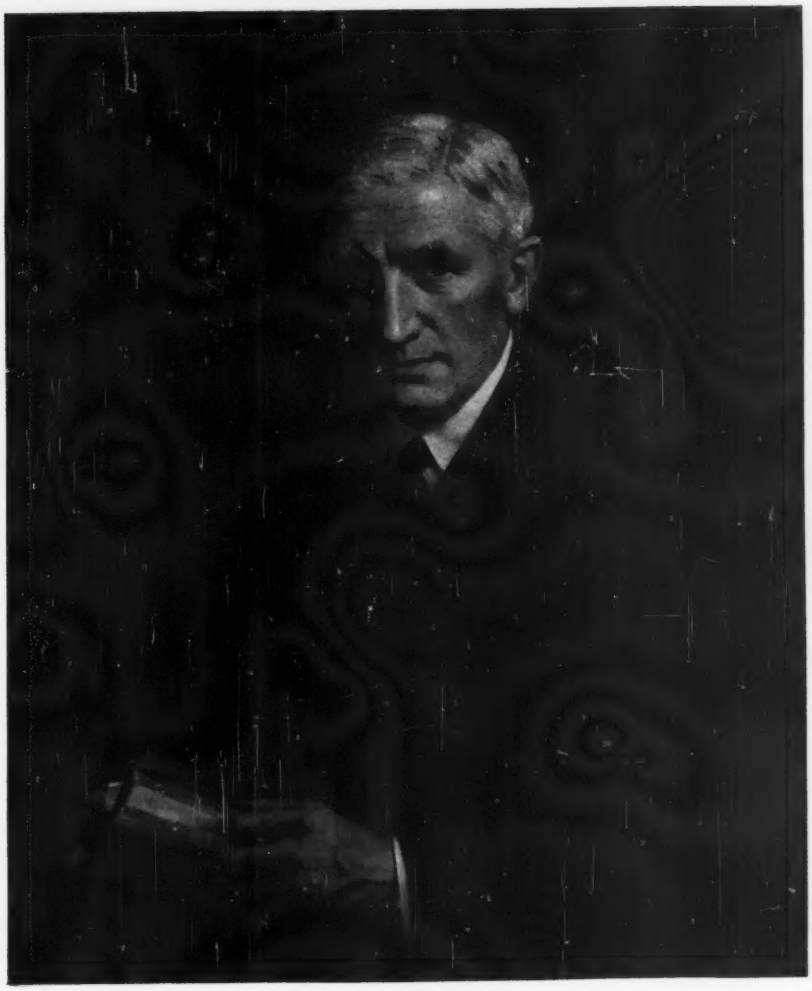
For some time he acted as assistant to Victor Horsley, and this association gave him the opportunity to develop his interest in the surgery of the central nervous system. Following the principles which he habitually taught, he thought about the problems which the skull and its contents presented for several years before he wrote his contribution on the "Scalp, Skull, and Brain" to Choyce's *System of Surgery*, and his short but authoritative paper on "Chronic Subdural Hæmorrhage" in the *British Journal of Surgery*, in 1914.

He became Assistant Surgeon to University College Hospital in 1906, by which time he had already clarified in his mind the principles upon which the operative treatment of carcinoma of the tongue should be based. His success in the practical application of these principles led him to extend them to the treatment of malignant disease of the pharynx, which at that time in this country was a neglected field of surgery. In his development of the technique of lateral pharyngotomy he was able to employ his exact knowledge of anatomy and his delicate skill in dissection to great advantage; but what attracted him most was the clinical recognition of the exact site and earlier phases of the disease, and the problem of surmounting the dangers of infection in the vulnerable tissues of the neck. As usual, it was only after several years of experience of his subject that his first paper, "On Certain Clinically Obscure Malignant Tumours of the Naso-pharyngeal Wall", appeared in the *British Medical Journal* towards the end of 1911. His Hunterian Lectures on "The Principles and Technique of the Operative Treatment of Malignant Disease of the Mouth and Pharynx", in 1913, established his unquestioned authority in this branch of surgery and brought surgeons of all nations to see him work.

His other chief surgical interest lay in the treatment of thyrotoxicosis, of which he was an acknowledged master but about which he wrote little. In later life he was greatly encouraged by his son's interest in this field.

The War of 1914–8 brought to him, as to others who remained at their posts, additional work in the care of the wounded, but he found time to write the results of his experience in the handling of wounds of the central and peripheral nervous system, and to add to his papers on the herd instinct a topical corollary in which he reviewed the reactions of the German people in the light of his thesis. The resulting volume was published in 1916 in London and New York, under the title of *Instincts of the Herd in Peace and War*.

During the decade which followed the War, Trotter's practice was limited only by his physical capacity to cope with it and by his refusal to allow his intellectual life to be submerged by the fatigue of routine. From 1924 until his death he took an active part on the Council of the Royal College of Surgeons, where his keen, analytical mind and mastery of the telling phrase were always at the service of progress. His selection as Hunterian Orator in 1932 called from him, in "The



WILFRED TROTTER

1872—1939

Commemoration of Great Men", the finest and most significant of the addresses of his maturity. His election to the Fellowship of the Royal Society in 1931 in recognition of his contributions to clinical surgery, was widely approved throughout the profession and brought him the greatest pleasure of any of his many academic honours. Among these may be mentioned his election to the Medical Research Council and to the Presidency of the Association of Surgeons in 1932.

In 1928 Trotter's surgical skill had contributed to the recovery of his late Majesty King George V from grave danger, and four years later he was appointed Serjeant Surgeon to His Majesty.

Many men besides Trotter have felt the claims and the enjoyment of hospital duties to be greater and more lasting than those of private practice, but there can be few others whose keenness of vision has enabled them to translate the wish into reality. Trotter had often discussed the project with his usual detachment, and in his later fifties he decided to devote the rest of his active years to the academic life. At the age of 63 he was invited to succeed C. C. Choyce in the Chair of Surgery at University College Hospital, and for the next two years, in spite of failing health, his energies ran smoothly along an ever-widening channel. In this short time he was able to sketch in the minds of his assistants an outline of his surgical ideals, and, in doing so, taught them many things besides technique.

Thinking was Trotter's hobby, with gardening as the opportunity for its unhindered exercise. The practice of his craft as a surgeon gave him real pleasure and he deliberately selected the more difficult fields of operation to obtain the stimulus of their exacting demands. During the early years of the century, when his ideas and surgical interests were taking the shape of their future maturity, the aseptic method had but recently established itself and the technique of its practice was still in process of evolution. An apologetic trust in antiseptics still lingered among the crevices of seniority. In this atmosphere it was natural that his aspiring ambition should be drawn towards surgical fields where errors of technique were inevitably punished. The relatively slight interest which he took in the abdomen as a field for surgery was determined by the knowledge that the peritoneum would draw an adherent veil over the difference between perfection and adequacy in surgical technique.

He used the English language as he used his hands, with a delicate precision born of constant striving towards control of his medium. While he was in the early thirties the conscious endeavour to find the exact word which would express his meaning was still occasionally recognizable in his speech, but as he grew older his command of word and phrase became so complete that the right word appeared to rise unsought. Though gifted with a mordant wit and an eye alert for hypocrisy, sloth, and the enunciation of platitudes, he was a man of rare and gentle courtesy. He could maintain attention to the opinions of others even when those opinions were trivial or were founded on ideas which he had long outgrown. Such was his kindness and tolerance that he could seldom bring disillusion to the ideals of honest stupidity, and many who were unable to follow even the simpler workings of his mind have added to their admiration of the man a claim that he was in some special way a close, because an understanding, friend.

His way of living was simple and he travelled little in the flesh, but he was a wide reader and a man of high intellectual courage. The expressed approval of his equals gave him keen pleasure, but it was entirely in keeping with his character that on more than one occasion he refused public recognition of his merits.

Few could speak with Trotter for long without becoming aware of the fundamental modesty of his mind. Not that he was uncritical of convention, or in any sense patient with those whose attention to a problem flagged because they could not solve it by a ready formula. He was often contemptuous of the too obvious introduction of personal emotion into the discussion of an intellectual problem. His modesty was, no doubt, inborn, but it was developed throughout his life by a conscious and deliberate striving towards impersonal thought. It might well be said of him as he said of Hunter, " his greatness was revealed . . . by his example in the use of the scientific method—faithfully, in purity of heart, and with utter devotion to truth ".

COLLES'S FRACTURE*

By J. H. MAYER

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It is an unfortunate fact that Colles's fracture has come to be regarded as a relatively easy injury to treat, and one that may be expected to give almost uniformly good results: unfortunate, because such happy optimism is by no means justified by a critical examination of the end-results, and because the 15 or 20 per cent of imperfect or frankly poor results with which some workers appear to be satisfied constitute a formidable number of permanently damaged wrists in this country every year.

Platt[9] (1932) reported a series with 83 per cent perfect and excellent late results, 14 per cent showing some defect, and 3 per cent poor, while three years earlier Edwards and Clayton[3] had published a series of Colles's and 'backfire' fractures in which, in reply to a questionnaire, 73 per cent of patients claimed perfect late results, 23 per cent owned to minor complaints, and 4 per cent were bad. On the other hand, Grasby and Trick[4] (1929) concluded from a short series that the late functional results after displaced Colles's fractures are invariably unsatisfactory, and, while this may be an exaggeration, a review in 1937 of the cases treated at Lambrinudi's Fracture Clinic at Guy's Hospital during the previous year showed that they left much to be desired.

It is certainly true that function may be perfect in the presence of slight persistent displacement, but perfect anatomical restoration is essential to consistently good functional results. In this review, it soon became apparent that the final anatomical result was satisfactory in considerably less than half of all displaced cases, and a careful analysis of serial radiographs in the unsatisfactory cases suggested that the failures were due to inadequate splintage after reduction. This is quite contrary to the teaching of Sir Robert Jones[6] (1929), Bankart[1] (1929), and other authorities, who have published the opinion that the method of splintage is of no importance if reduction has been perfect, and that redisplacement does not occur under these circumstances; but the radiological evidence of this fact is supported by experimental fractures which I have produced in dissection specimens, and by the improvement in results effected by a new method of splintage suggested by Lambrinudi[7] (1938).

CAUSES OF FAILURE OF TREATMENT

It became apparent, when analysing the cases treated up to November, 1937, that there was in the great majority an additional element in the original displacement to those ordinarily described, namely, a rotation of the lower radial fragment on its long axis, in a direction of supination. This 'supination-twist' carries the lower radial fragment round, and sometimes away from, the head of the ulna, and away from the pronated upper radial fragment.

Cotton[2] emphasized the importance of such a 'rotary displacement in a direction of supination' fifteen years ago, but it still passes unrecognized, largely,

* Based on a paper read before the British Orthopædic Association Spring Meeting, 1939, at Oswestry.

it seems, from lack of data in X-ray interpretation. This difficulty has been overcome by X-raying various separate displacements in experimental fractures, and it has become clear from these studies that, although the supination-twist may be easily mistaken for other possible displacements, certain criteria can be laid down by which its existence and severity may always be recognized radiologically.

The evidence so collected suggests that a supination-twist is an almost constant part of the deformity in true Colles's fractures, and if this is so, it is essential to pronate the lower fragment during reduction, as in the method of the late Sir Robert Jones. But it is surely irrational to reduce in this way and yet to splint the wrist without also controlling pronation-supination movements; and, since full pronation is necessary to bring the lower fragment into alinement with the upper, this is the position in which splintage must be maintained to prevent redisplacement. Sir Robert Jones's twisted gutter splints were designed to prevent supination of the lower fragment, but, as in all fractures, adequate control can be maintained only by fixing the joints both above and below the fracture, and it is in order to prevent rotary movement that the flexed elbow should be included in the plaster.

Shortly after this method had been instituted at Guy's Hospital in November, 1937, Taylor and Parsons[10] (1938) advised precisely the same treatment for cases in which the inferior radio-ulnar joint is damaged, but we accept far wider indications for its use.

EXPERIMENTAL FRACTURES

Ordinary dissecting-room specimens were used, and so far as possible the soft tissues were left undisturbed.

In the first stage, the radius was divided at its typical site with a Gigli saw, with the forearm pronated as at the moment of production of a true Colles's fracture. The result of attempting to produce a typical Colles's displacement now, by backward pressure on the hand, was as follows :—

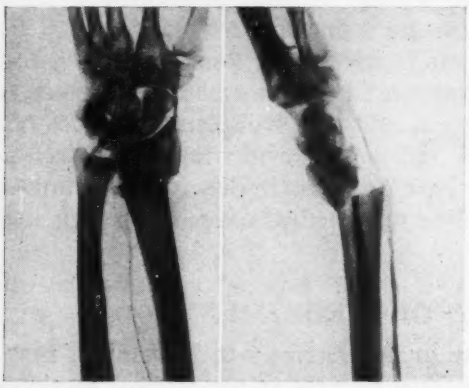


FIG. 448.—Experimental fracture, first stage, showing the maximum displacement possible with division of the radius only; moderate backward angulation and a severe supination-twist.

a. A slight backward angulation occurred, but this remained moderate in amount even with strong pressure on the hand.

b. The most striking displacement was rotation of the lower radial fragment round the head of the ulna, in a direction of supination.

c. There was no displacement in any other direction. It proved quite impossible to produce either true backward displacement or radial deviation of the lower radial fragment.

The radiograph of this displacement is shown in *Fig.* 448. Strong backward pressure was exerted on the hand as this radiograph was taken, and it may be accepted that this is the maximum deformity that can be produced at this stage. Moderate backward angulation is visible in the lateral view, and also :—

1. In the lateral view, apparent backward displacement of the lower fragment.

2. In the antero-posterior radiograph apparent radial deviation of the lower fragment, which appears to overlap the upper fragment on its outer side, and to be slightly broadened in comparison with it.

No such backward or radial displacement was in fact present, and these appearances are typical of a severe supination-twist. The diagram (*Fig.* 449) shows how such appearances may be due to this cause; and they can be due to none other because no other displacement was present when *Fig.* 448 was taken.

Exploration of the wrist at this stage in the experiments showed that the axis of the supination-twist was the attachment of the triangular fibrocartilage of the

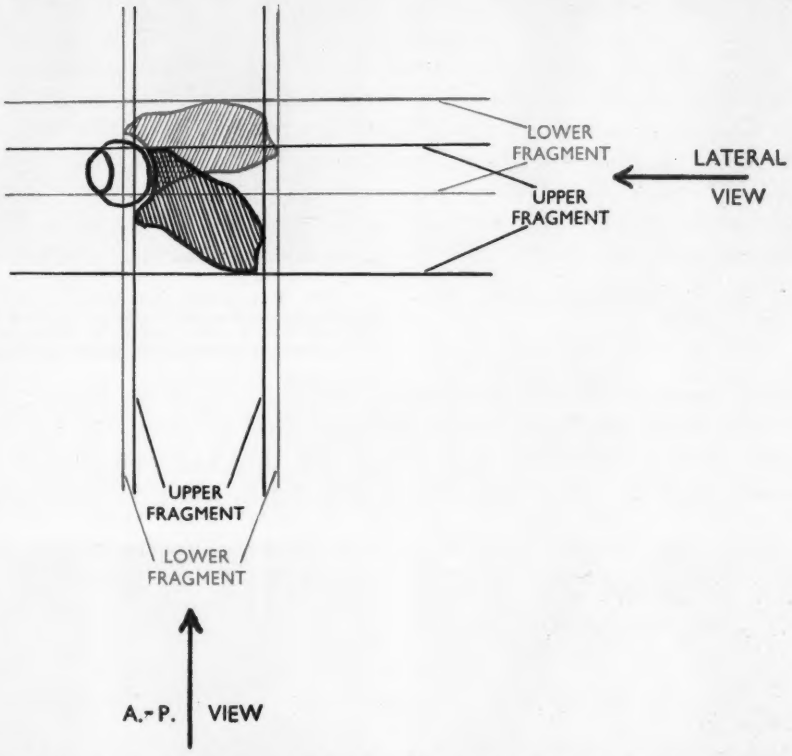


FIG. 449.—Diagram to show how the radiographic appearances of a supination-twist are produced. Obvious variations may occur according to the exact direction of the X rays.

inferior radio-ulnar joint near the base of the ulnar styloid process. As the hand was forced backwards and the radius angulated slightly, the anterior fibres of the fibrocartilage were put 'on the stretch,' and the lower fragment, prevented by this tension from backward displacement or further backward angulation, was swung round into supination by the fibrocartilage.

X-ray findings exactly similar to those in *Fig.* 448 are not uncommon in true Colles's fractures. Such a case is shown in *Fig.* 450, while another common appearance is that illustrated in *Fig.* 451, where the lower fragment appears in the antero-posterior view to overlap the upper on both sides, and to be considerably broadened, again with apparent backward displacement in the lateral view. Such differences in the radiographic appearances of a supination-twist are accounted for by slight variations in the direction of the radiographs, and by the varying severity of the twist.

If now in the experimental fractures the styloid process of the ulna were divided at its base, the complete Colles's displacement could be produced without difficulty (*Fig.* 452).

Dissection showed that the fibrocartilage is attached to the proximal part of the ulnar styloid process rather than to the fossa at its base, mainly by anterior

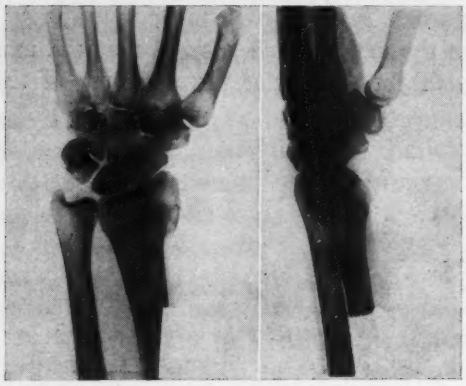


FIG. 450.—Colles's fracture with a marked supination-twist (cf. *Fig.* 448).

FIG. 451.—Radiograph with the lower fragment apparently overlapping the upper on both sides in the antero-posterior view, due to a supination-twist, and with moderate backward angulation (cf. *Figs.* 448, 449).

and posterior bands of fibres which become continuous with the thick anterior and posterior borders of the fibrocartilage diverging from its apex. By fracturing the base of the styloid process, the fibrocartilage was detached with it from the head of the ulna, so now the lower fragments of both bones, joined by the fibrocartilage,

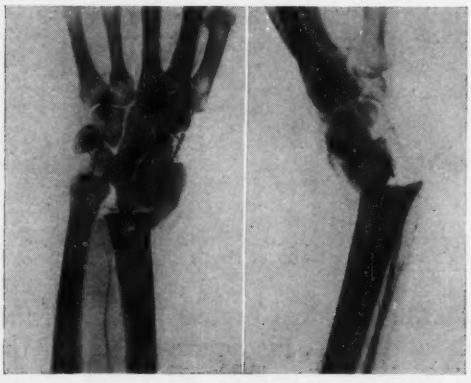


FIG. 452.—Experimental fracture, second stage. The same specimen as *Fig.* 448, showing the increased deformity allowed by division of the base of the styloid process of the ulna.

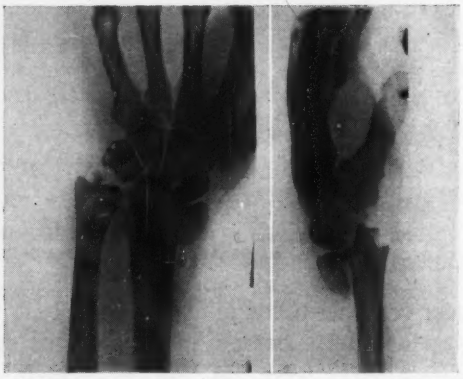


FIG. 453.—Colles's fracture with displacement corresponding with that shown in *Fig.* 452.

could be displaced as an entirely separate unit, and the lower radial fragment was no longer pivoted round the head of the ulna. As the complete Colles's displacement was produced, the ulnar styloid was pulled backwards and slightly towards the radial side by the fibrocartilage.

Fig. 453 shows a true Colles's fracture with an almost identical displacement with that of the experimental fracture in *Fig.* 452. A supination-twist is again clearly visible.

If at this stage in the experiments the fracture at the base of the ulnar styloid was replaced by division of the fibrocartilage itself, or its detachment from either of its bony attachments, the result was the same in allowing the previously partial bony deformity to be completed.

MECHANISM OF COLLES'S FRACTURE

On the basis of these experiments the following suggestions are made.

As the pronated hand strikes the ground the radius is fractured and the residual force tends to drive the lower fragment backwards. Moderate backward angulation results, but the tendency to further backward displacement produces tension on the fibrocartilage, and particularly on its anterior fibres, with the result that the lower fragment is pivoted round the head of the ulna in a direction of supination.

With a moderate force acting, this will be the total displacement, which might be termed a 'first degree' fracture. The intact attachment of the fibrocartilage to the head of the ulna prevents more than moderate angulation, and precludes true backward displacement or radial deviation.

FIG. 454.—Spiral fracture of the neck of the ulna in a Colles's fracture with severe displacement.

If the force of the injury is sufficiently great, however, the sudden tension on the fibrocartilage may result in an avulsion-fracture of either of its bony attachments, usually the ulnar styloid, but sometimes the radial attachment; a third, rather hypothetical, possibility is rupture of the fibrocartilage itself, but this has not been diagnosed in any case of the present series. Only when the lower radial fragment is set free in one of these ways from the head of the ulna can the full Colles's displacement occur, and these cases might well be defined as 'second degree' fractures.

It seems from the experimental fractures that a fracture at or near the base of the ulnar styloid is always the result of avulsion by the fibrocartilage, and not by the ulnar collateral ligament of the wrist-joint. Indeed, it is extremely difficult to see, in dissected specimens, how this ligament can possibly be put under tension at any moment in the production of a typical Colles's fracture.

Fracture of the neck of the ulna may replace or coincide with a fracture at the base of the ulnar styloid, and I suggest that this also is a traction-fracture produced by the fibrocartilage. In all such cases in the present series the ulnar fracture was a spiral one, as would be expected if it were produced in this way (*Fig.* 454).

Again, radial avulsion fractures often consist only of the anterior portion of the border to which the fibrocartilage is attached (*Fig.* 455), which is to be expected if the anterior part of the fibrocartilage is under the greatest tension. On the other hand, a relatively large part of the lower radial fragment may remain attached to the fibrocartilage, producing a T-shaped radial fracture in which the lateral

fragment is set free but the medial retains its attachment to the head of the ulna (*Fig.* 456).

More severe angulation than that illustrated in *Fig.* 448 can of course occur only in one of the varieties of 'second degree' fracture, and for a diagnosis of ruptured fibrocartilage one would require the presence of greater angulation than this without evidence of any avulsion-fracture.

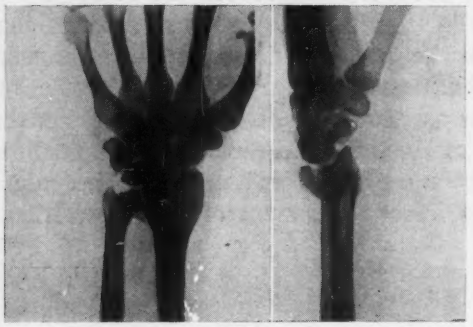


FIG. 455.—Colles's fracture with avulsion of the anterior part of the radial attachment of the fibrocartilage.

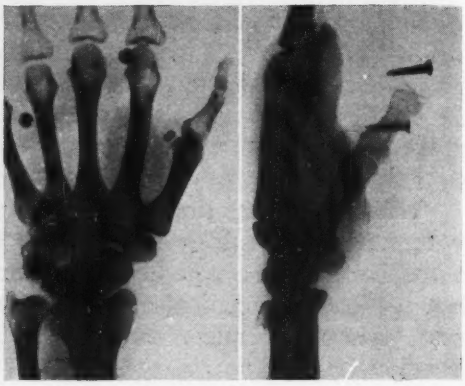


FIG. 456.—T-shaped fracture of the lower end of the radius.

THE INFERIOR RADIO-ULNAR JOINT

The significance of damage to this joint was suggested during the early stages of this investigation by the frequency of pain on the ulnar side of the wrist as a late symptom, and by the difficulty in regaining full pronation-supination movements in our early cases.

In this connexion it is interesting to recall that limitation of pronation-supination movements following 'luxation of the wrist' was emphasized, 88 years before Colles's accurate description of the fracture, by Petit[8] (1726), who suggested that it was due to damage of the triangular fibrocartilage.

It is now clear from my anatomical studies that, in 'first degree' fractures, the inferior radio-ulnar joint remains intact in all except the occasional case with the fracture line involving the ulnar-joint surface of the radius. In 'second degree' fractures, however, the surfaces of this joint are nearly always damaged, in one of the following ways :—

a. Avulsion of the ulnar styloid by the fibrocartilage.

b. Avulsion of its radial attachment by the fibrocartilage.

c. Possibly rupture of the fibrocartilage itself.

The two exceptions in which the inferior radio-ulnar joint remains intact on account of the very size of the fragments avulsed, are: (1) Fracture of the neck of the ulna; (2) T-shaped fractures of the lower end of the radius.

There is no general agreement on the probability of subsequent osteo-arthritis of this joint: Platt holds that it is a very rare sequela of Colles's fracture, whereas Watson-Jones[5] (1929) believes it to be the most usual cause of persistent pain and disability. One of the joint surfaces being formed by the fibrocartilage, it cannot be expected that all cases of osteo-arthritis will be apparent radiologically, but by

analogy with other joints it is reasonable to assume that arthritis is a likely sequel to permanent damage of the articulating surfaces.

It is with a view to maintaining correct anatomical restoration of these that Taylor and Parsons advise splintage in full pronation, but they believe such damage to be less common than our cases suggest, and I consider that other types of Colles's fracture also are best treated by this method.

INDICATIONS FOR SPLINTAGE IN FULL PRONATION

As a result of this anatomical investigation and of experience in the clinical use of the method, it may be said that Colles's fractures are best splinted in full pronation under either of the following circumstances :—

a. Evidence of a supination-twist in the original displacement.

b. Evidence of damage to the inferior radio-ulnar joint, as determined by the criteria enumerated above.

The only exception to these indications is the case, showing a supination-twist, with a fracture of the neck of the ulna, as here the stability of the fulcrum is lost: this type is best splinted after reduction in about the mid-prone position, with the elbow included in the plaster to control rotary movements.

Of the cases analysed later in this paper, 84 per cent obeyed these indications for splintage in full pronation.

REDUCTION AND SPLINTAGE

Whatever method of reduction be used, a final powerful twist of the lower fragment into pronation is absolutely essential in these cases. Reduction of the experimental fractures by a combined manipulation of traction, forward angulation, ulnar deviation and pronation was consistently successful in producing a perfect position, and the ulnar styloid could be seen to fall back into its proper alinement in those cases where it had been fractured. Perfect bony union of a fractured ulnar styloid has been obtained in a proportion of the clinical cases treated in full pronation.

General or local anæsthesia may be used for reduction, but muscular relaxation must be complete. To achieve this by local anæsthesia, it may be necessary to inject the ulnar fracture separately.

Immediately after reduction a skin-tight plaster is applied with the forearm and hand fully pronated, the wrist ulnar-deviated and slightly palmar-flexed, and the elbow flexed to a right-angle, extending from the metacarpal necks to about the middle of the humerus (*Fig*. 457). Ulnar deviation is maintained in an attempt to prevent radial shortening, an unfortunate occurrence which it has been found most difficult to avoid. In fact, it seems that this is an almost inevitable sequela in fractures with marked comminution, whatever method of treatment is employed, short of continuous traction. Particular care is taken in moulding round the lower end of the radius, and the plaster is not bivalved or otherwise disturbed. Great care is of course necessary in the application of the plaster and the subsequent treatment to avoid circulatory disturbances, but by instituting active finger and thumb exercises under daily supervision from the beginning of treatment we have escaped such untoward accidents.

We have left such plasters for the full period of five weeks' splintage, but this leads to unnecessary difficulty in the subsequent mobilization, and we now maintain full pronation for three weeks, the period during which redisplacement is otherwise likely to occur, and then change to a short plaster, with straight wrist and not including the elbow, for a further two weeks while consolidation takes place.

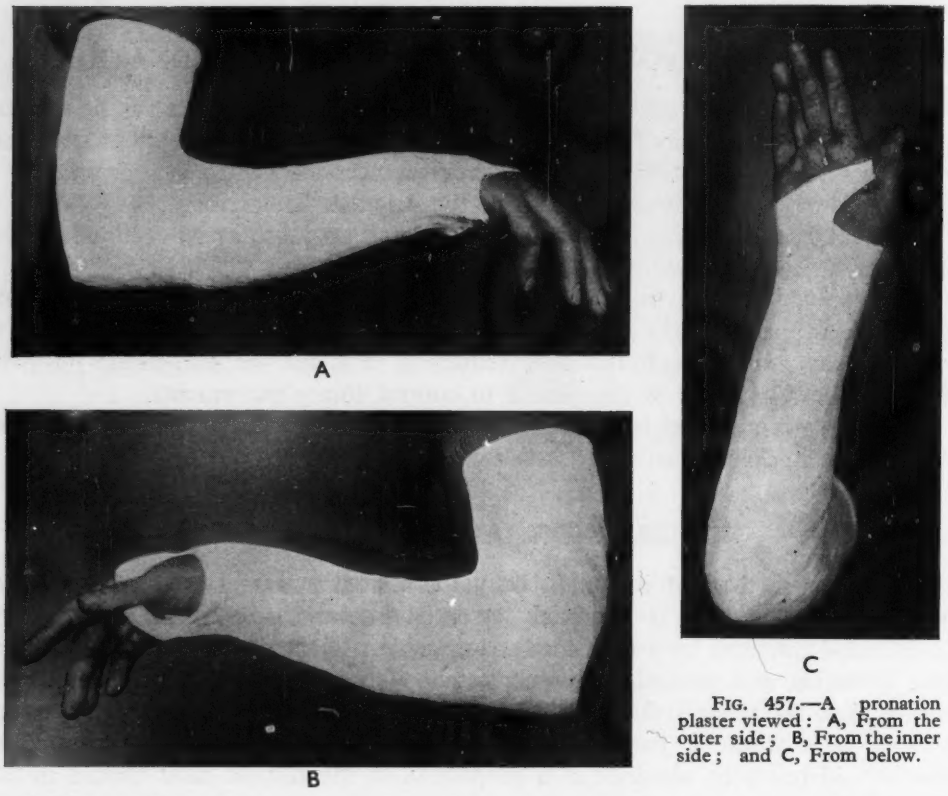


FIG. 457.—A pronation plaster viewed: A, From the outer side; B, From the inner side; and C, From below.

We have found it useless to apply a pronation plaster without anæsthesia, even if adequate reduction has been performed already, as full pronation can never be obtained under such circumstances, and in this series redisplacements were just as common in cases fixed in partial pronation as in the various other methods tried. These other methods include skin-tight plasters not including the elbow, with the wrist fixed (*a*) straight, (*b*) in partial palmar-flexion, (*c*) in full palmar-flexion, (*d*) in full ulnar deviation, and (*e*) with a pronation-twist; and also skin-tight plasters incorporating the elbow in supination. None of these methods reduced the incidence of redisplacement, so for the purposes of this review all except the full pronation technique have been classed together as 'other methods'.

CASES REVIEWED

Sixty-three displaced Colles's fractures treated between November, 1936, and December, 1938, have been reviewed. These are unselected and consecutive cases, except that only those are included about which full details of treatment, including a complete series of radiographs, were available for re-examination. Those in

which, because of advanced age or other considerations, reduction was not undertaken, are excluded.

Of these cases 43 fall under the heading 'second degree' fractures, as follows:—

a. Fractures at or near the base of the ulnar styloid (34 cases); 3 of these also had small radial avulsion-fractures and 4 had T-shaped radial fractures.

b. Fractures of the neck of the ulna (5 cases); 2 of these also had small radial avulsion-fractures.

c. Avulsion of the radial attachment of the fibrocartilage alone (4 cases).

Groups (*a*) and (*c*) and 2 cases in group (*b*) had evidence of damaged inferior radio-ulnar joints—a total of 40 cases.

Of the 63 cases, 53 showed a supination-twist in the original radiograph, including 38 'second degree' and 15 of the 20 'first degree' fractures.

It is noteworthy that only 8 of the 63 cases had fractures at or near the tip of the ulnar styloid.

According to the indications laid down above, 53 of these cases should have been treated in full pronation, as follows: (*a*) Forty-eight cases with a supination-twist (i.e., all those with a supination-twist except 5 with fractures of the neck of the ulna). (*b*) Five cases with damaged inferior radio-ulnar joints, but no obvious supination-twist.

Only 20 cases were so treated because the method was instituted midway through the series, and because only the most severe displacements were at first selected for its trial.

ANATOMICAL RESULTS

Serial radiographs of all these cases have been reviewed, and these confirm the fact that redisplacement may occur at any time during the first two or three weeks.

Of the 63 cases 5 were re-reduced after slipping badly during the first two weeks of splintage, and the results of their re-reduction are included in the tables that follow, in addition to those of their original reduction, making the total 68 reductions.

The cases have been analysed (*Table I*) in two groups—those splinted in full pronation and those treated by other methods. Rigid criteria of perfection have been maintained throughout, and any imperfections of radial alinement, including persistent supination-twist and radial shortening, have been classified as 'good' but not 'perfect'.

Table I.—Results of Treatment in Colles's Fracture

Reduction	Redisplacement	Pronation Plasters	Other Plasters	Result
Perfect	None	7 (64 per cent)	5 (24 per cent)	Perfect
	Slight	4 (36 per cent)	10 (48 per cent)	Good
	Severe	0	6 (28 per cent)	Fair
Imperfect	None	7 (77 per cent)	5 (20 per cent)	Good
	Slight	2 (23 per cent)	15 (60 per cent)	Fair
	Severe	0	5 (20 per cent)	Bad
Bad	None	0	0	
	Worse	0	2 (100 per cent)	Bad
Total Cases		20	48	

Surprisingly few perfect reductions were achieved, judging by these standards, but there was a larger proportion of these in the pronation cases than in the others, due no doubt to the strong pronation-twist employed during reduction. Four of the pronation cases were slightly 'over-pronated', but this 'over-reduction' did not appear to interfere with their subsequent functional recovery. It is almost impossible to 'over-reduce' in other planes.

The anatomical end-results are summarized in *Table II*, from which it will be seen that 90 per cent of the pronation cases gave satisfactory results, and none were worse than 'fair', whereas only 41 per cent of those treated by other methods were satisfactory and 15 per cent were frankly 'bad'. These results were achieved although we were only developing the technique at the time, and although the pronation cases were specially selected as being particularly liable to redisplacement.

Many of the slighter redisplacements in both groups consist only of radial shortening, but only 6 out of 20 pronation cases slipped at all during splintage, as against 38 out of 48 others.

Table II.—Anatomical End-results in Colles's Fracture

Anatomical Result		Pronation Plasters	Other Plasters
Satisfactory	Perfect	7 (35 per cent)	5 (10 per cent)
	Good	11 (55 per cent)	15 (31 per cent)
Unsatisfactory	Fair	2 (10 per cent)	21 (44 per cent)
	Bad	0	7 (15 per cent)
Total Cases		20	48

The results of treatment in full pronation in three typical cases are shown in *Figs.* 458–460. That shown in *Fig.* 458 was the first patient in whom this method was applied. The original displacement (*Fig.* 458, A) was extreme, with very marked radial angulation and the ulnar styloid fractured at its base and markedly displaced backwards. Initial reduction (*Fig.* 458, B) was good, and a skin-tight plaster was applied, including the flexed elbow, in a position of supination, but one week later the fracture had slipped to the position shown in *Fig.* 458, C, and re-reduction was performed. A pronation plaster was then applied (*Fig.* 458, D), and this was removed five weeks later, when the final radiograph (*Fig.* 458, E) was taken, showing union in perfect position, including fusion of the ulnar styloid. The patient, a man aged 42, had full painless movement when last seen.

Fig. 459, A, shows a case (female, aged 62) with a marked supination-twist, severe angulation, and avulsion of part of the radial attachment of the fibrocartilage. The final radiograph, after two weeks in full pronation and three weeks in a short plaster, is shown in *Fig.* 459, B, and the wrist, re-examined nine months after the injury was strong and painless, with supination a little short of full, but still improving.

Fig. 460, A, shows another 'second degree' fracture, with avulsion of the ulnar styloid. This is one of the cases over-pronated during reduction, resulting in apparent forward displacement of the lower radial fragment (*Fig.* 460, B), but the patient, a woman aged 47, suffered no worse disability than slightly diminished supination when last examined five months after her injury.

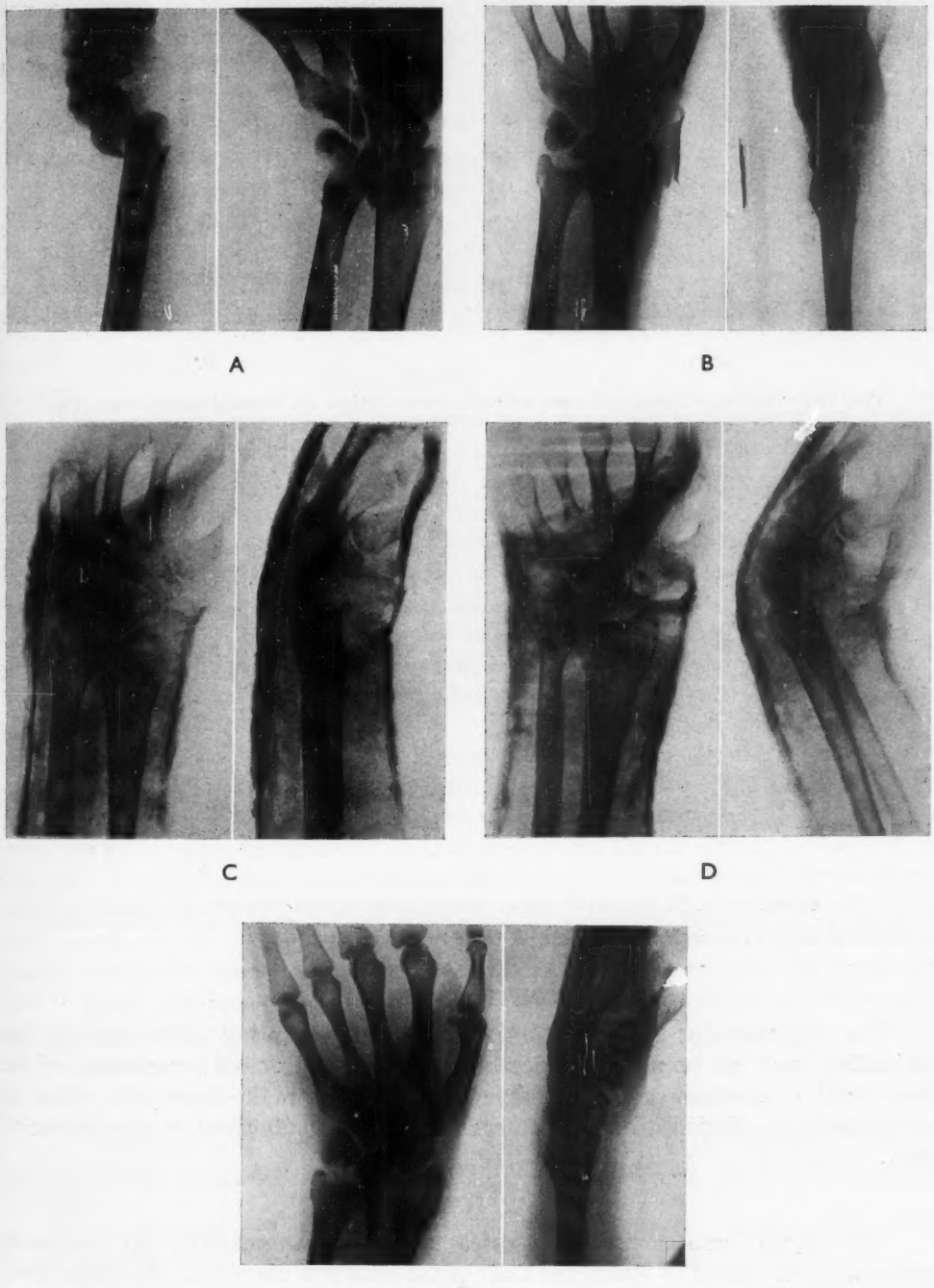
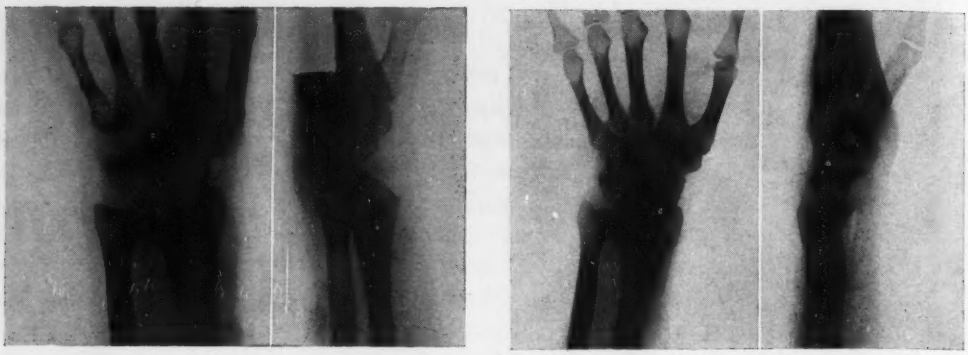
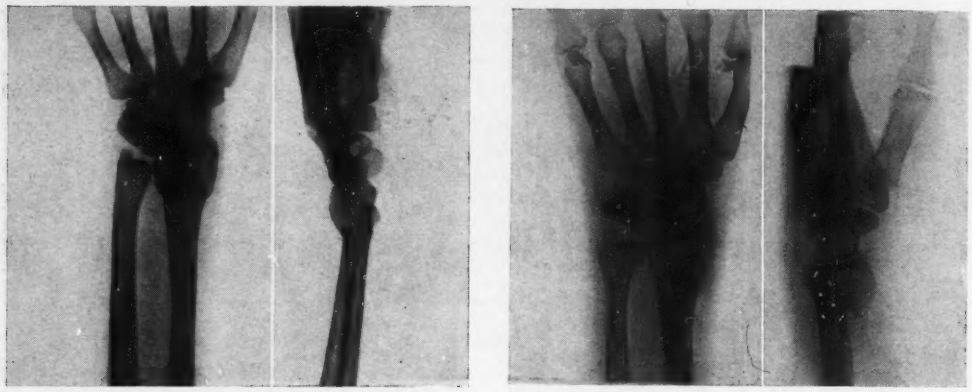


FIG. 458.—Radiographs of a case (E. M., male, 42 years) in which plaster in full pronation resulted in union in perfect position, after the failure of splintage in supination. A, Original radiograph; B, Same case, initial reduction, position good; C, Redisplacement one week later, in plaster incorporating the elbow, in supination; D, After re-reduction, plastered in full pronation with partial palmar-flexion; E, Final radiograph. Both radial and ulnar fractures have united in perfect position.



A B

FIG. 459.—Case M. R., female, 62 years, treated in full pronation. A, Original displacement, with partial avulsion of the ulnar joint surface of the radius; B, Final radiograph of the same case.



A B

FIG. 460.—Case M. B., female, 47 years, 'over-pronated' during reduction and splinted in full pronation; A, Radiograph showing the initial deformity; B, Final radiograph showing apparent forward displacement of the lower radial fragment produced by 'over-pronation'.

COSMETIC RESULTS

The improvement in cosmetic results from preventing recurrence of the supination-twist has been very noticeable. The normal dorsal prominence of the ulnar head is regained and the laxity of the inferior radio-ulnar joint which is sometimes seen after displaced fractures has not been observed in the pronation cases.

COMPLICATIONS

One of the earliest pronation cases had two manipulations for persistent stiffness, at the second of which the shaft of the ulna was fractured. No other cases have needed manipulation.

Delayed rupture of the extensor pollicis brevis tendon occurred in one pronation case two months after the injury, and this was successfully treated by Mr. Lambrinudi by bisection of the abductor pollicis longus tendon and transplantation of one half into the ruptured tendon.

One case treated in a short plaster suffered the commoner belated rupture of the extensor pollicis longus tendon eight months after the fracture, but refused operation.

DURATION OF TREATMENT

The pronation cases take a little longer to mobilize after removal of the plaster than the others. If the three cases with complications noted above are excluded, the average period off work in this series was 10 weeks in the pronation cases and 8½ weeks in the others, while the average total duration of treatment was 16 weeks in the pronation cases and 11 weeks in the others, the additional time being occupied by attendance from two to four times a week for mobilization exercises.

FUNCTIONAL RESULTS

The functional results were reviewed in April, 1939. Details were obtained in 48 of our cases, mostly by personal examination and the remainder by written questionnaire.

The functional results were found to correspond fairly closely with the anatomical, and all cases with severe residual deformity had limitation of movement roughly in accordance with their anatomical defects both in degree and in direction. Of the remainder there were some with full range in spite of slight residual displacements, and a few without obvious skeletal cause for their limitation.

Some loss of normal movement was commonly found in patients making no subjective complaint, which suggests that figures based upon written replies are of somewhat doubtful value.

Pain or 'weakness' was present in exactly the same proportions of our two groups—7 out of 16 pronation cases and 14 out of 32 others followed up. There was limitation of movement in 6 of the 16 pronation cases, but in 16 of the 32 other cases, so this was already less common in the former group, in spite of the fact that the pronation cases had all occurred during the second year of the series, and represented all the worst displacements attending for treatment during that time, whereas the others were a 2-year series and included only the slighter displacements during the second year. The majority of recent cases were still improving, and it seems reasonable to assume that the ultimate results in the pronation cases will be considerably better than in the others.

SUMMARY

1. The results of Colles's fractures treated by present methods are extremely disappointing, redisplacement being exceedingly common even after perfect reduction.

2. An essential part of the displacement in most cases is a rotation of the lower radial fragment into supination, but this usually passes unrecognized.

3. The radiological appearances of such a 'supination-twist' are characteristic, and have been enumerated by X-raying experimental fractures in dissected specimens.

4. The triangular fibrocartilage of the inferior radio-ulnar joint plays a vital part in the mechanism of the fracture, and the inferior radio-ulnar joint is probably damaged in the majority of cases. Such damage may be recognized radiologically, and its proper repair must receive due attention.

5. Splintage in full pronation, with the flexed elbow included in the plaster, has proved to be the method of choice as a guard against redisplacement in all cases with a supination-twist and in all with damaged inferior radio-ulnar joints, with the single exception of those with a fracture of the neck of the ulna.

6. The anatomical and functional results in a short series so treated are considerably better than in a consecutive series treated by other methods, and it is hoped that this method will be afforded a more extensive trial.

I should like to conclude by thanking Mr. C. Lambrinudi, to whom belongs the credit of having instituted this method of treatment, for permission to use his patients in this investigation, and for his helpful advice and criticism in the preparation of this paper.

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A FURTHER REVIEW OF THE INTERINNOMINO-ABDOMINAL OPERATION: ELEVEN PERSONAL CASES

By GORDON GORDON-TAYLOR
SURGEON REAR-ADMIRAL

In a previous number of the British Journal of Surgery, Mr. Philip Wiles and the author made a communication upon the interinnomino-abdominal amputation (hindquarter amputation), which was based upon an experience of 5 cases in which they had been associated in the performance of this operation. Six additional operations of this kind have now been carried out by the writer, and in the light of this further experience it may not be untimely to review not only the details of the technique which were then suggested, but the end-results of the cases which have been subjected to this very severe mutilation.

The publication of the paper of 1935 at least appears to have stimulated other British surgeons to take an interest in the operation, and the surgical enterprise and skill of my friends have been rewarded by an almost unvarying record of surgical successes. In Britain, Harry Platt, Grey Turner, W. H. Ogilvie, and Duncan Fitzwilliams have all secured the operative recovery of their patients, and some of these surgeons have had more than one successful case; recent successes have also been recorded in the Dominions by Harris, of Toronto, and by McCaw, of Auckland.

In the former communication to this Journal, Philip Wiles went to immense trouble to collect the cases of hindquarter amputation recorded up to 1934; the operation had then been done about 79 times, with 46 deaths—i.e., a mortality-rate of 59·5 per cent—but it was suggested that this mortality-rate probably underestimated the real gravity of the operation.

A personal experience of 11 interinnomino-abdominal amputations is unique, and it is disappointing to find that familiarity with the operation and the greater perfection of the methods of intravenous infusion and transfusion therapy have not resulted in any appreciable lowering of the operative mortality in the second series of personal cases, when comparison is made with those published in 1935. Of the first 5 personal cases, 3 recovered from the operation, while 4 out of the last 6 have survived. It would appear that the magnitude of the operation is one which inevitably carries a mortality of about 1 in 3 and that any very great improvement is hardly to be expected.

PERSONAL CASES

Cases 1–5 were given in detail in the British Journal of Surgery, 1935, 22, p. 672.

Case 1.—**Sarcoma of upper end of femur with involvement of innominate bone.**

Operation, July 27, 1922. Death a few hours after operation.

Case 2.—**Osteoclastoma of innominate bone.**
Operation, February 4, 1929. Patient in excellent health 10½ years later.

Case 3.—**Enormous chondrosarcoma of innominate bone, overlapping upper end of femur and extending to costal arch.**
Operation, November 5, 1933. Death October, 1938, of recurrence.

Case 4.—**Sarcoma of pelvis.**
Operation, March 3, 1934. Death.

Case 5.—**Osteogenic sarcoma of upper end of femur.**
Operation, July 2, 1934. Recovery. Patient is said to have died of pneumonia less than a year after operation. It is difficult to exclude a thoracic metastasis.

Case 6.—**Spindle-cell sarcoma of muscles of Scarpa's triangle: operation; recovery.**
G. M., male, aged 17 years. Admitted from Malta with a large sarcoma of the muscles of Scarpa's triangle, which had been present for several months. The requirements of a really radical operation would have been ill satisfied by a disarticulation of the hip-joint, and arrangements for an interinnomino-abdominal operation were made.
OPERATION (June, 1935).—Dr. A. E. W. Idris gave stovaine and ether. Messrs. Philip Wiles, J. Leedham-Green, and Anthony Till helped me. A preliminary biopsy confirmed the malignant character of the neoplasm and operation was straightway proceeded with. Very considerable shock attended the operation. The tumour was a spindle-celled sarcoma. An excellent recovery followed the operation, and the patient is in good health nearly five years later, and a medical student at a London medical school.

Case 7.—**Osteolytic sarcoma of iliac portion of right innominate bone; pathological fracture of acetabulum: operation; death a few hours after operation.**
Florence C., aged 37, a diminutive woman of only 4 st. in weight, was admitted into my surgical unit at Middlesex Hospital for pain in the right hip which had been present for sixteen months; there was a lump over the posterior portion of the right iliac bone. The pain in the hip was especially severe at night, and kept her awake; the patient had been confined to bed for four months before admission. A great deal of weight had been lost in the past sixteen months. Movements of the hip-joint were limited and painful.
X-ray report: There is a large area of destruction in the wing of the right ilium and the margin of the sacrum is also involved. The appearances are those of a neoplasm, possibly an osteolytic sarcoma. A pathological fracture of the right acetabulum is present. No secondary growths in the thorax could be discerned on X-ray examination.
The patient was placed upon a high calorie diet for nearly 3 weeks, and a transfusion of blood was given four days before operation.
OPERATION (July 25, 1936).—Dr. A. E. W. Idris gave stovaine and ether. J. Leedham-Green and Brian Truscott helped me. The transfusion arrangements were in the hands of Mr. O. Lloyd-Davies. Right interinnomino-abdominal operation. Owing to the position of the growth, the division of the ilium from crest to sciatic notch was impracticable, and a disarticulation was practised at the sacro-iliac joint. A piece of the ala of the sacrum was removed to ensure an adequate extirpation of the growth. The operation was attended by severe shock, and the patient succumbed three hours after the operation had been completed.
POST-MORTEM EXAMINATION (Middlesex Hospital 149/1936): There was no evidence of any further new growth in the body.

Case 8.—**Spindle-celled sarcoma of muscles of Scarpa's triangle: operation; recovery.**
Florence W., aged 25, married, one child; admitted into my surgical unit, Middlesex Hospital, for a large swelling in the uppermost part of the left thigh. The diagnosis of a sarcoma was confidently made, but a preliminary biopsy was performed. The tumour proved to be of excessive vascularity and finally the femoral artery had to be ligated to control the hæmorrhage. Severe constitutional disturbance followed this surgical essay, and three weeks had to elapse before the patient was considered to be in a fit state to stand the severe ordeal of a hindquarter amputation.

OPERATION.—Dr. H. P. Crampton gave stovaine and ether. Messrs. Philip Wiles and Anthony Till helped me. The usual technique was followed, the posterior portion of the iliac portion of the innominate bone being sawn through from iliac crest to sacro-sciatic notch. The remaining auricular portion of the innominate bone was then removed by disarticulation at the sacro-iliac synchondrosis (*Fig.* 461).

The patient made a good recovery from the operation, and is alive and well 3 years after the amputation.

FIG. 461.—*Case* 8. Spindle-cell sarcoma of muscles of Scarpa's triangle. Amputation. Patient alive and well 2½ years later.

Case 9.—**Sarcoma of right innominate bone: operation; recovery.**

Ronald S., 14 years of age, was admitted under my care in Middlesex Hospital on August 2, 1938, for a large sarcoma of the right innominate bone.

OPERATION (August 10, 1938).—Dr. Crampton gave stovaine followed by ether. The usual technique was followed, and at the completion of the operation surgery appeared to have gone well beyond the limits of the tumour (*Fig.* 462.)

PROGRESS.—A good immediate recovery from the operation resulted, and the patient was discharged from hospital on October 2, 1938.

About five months afterwards the patient developed pain in his lumbar region, followed by sciatica and paralysis of the remaining limb. There seems little doubt that a secondary growth developed in his spine. Death took place six months after the amputation.

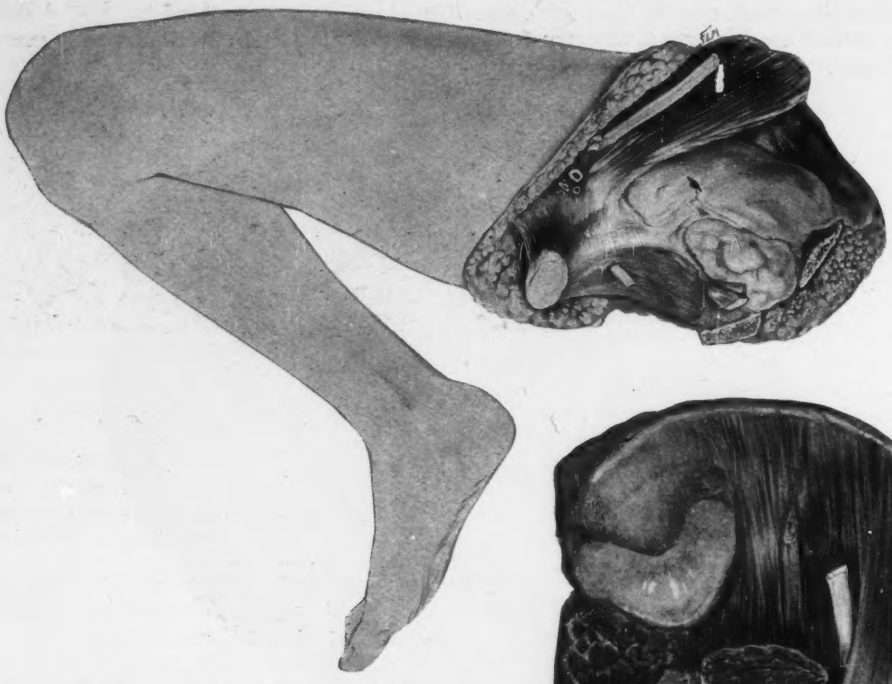

FIG. 462.—*Case* 9. Sarcoma of right innominate bone. Recovery from operation. Death of recurrence in spine six months later.

Case 10.—**Spindle-cell sarcoma of right psoas muscle: amputation; death.**

Charles O., aged 34, was admitted under my care in the Middlesex Hospital with a lump in the uppermost part of the right thigh; the swelling was also just palpable above Poupart's ligament. The characters of the tumour left little room for doubt as to its malignant character. Clinical and radiological investigation showed no evidence of thoracic secondary growths, and interinnomino-abdominal amputation was decided upon.

OPERATION (September, 1938).—Dr. H. G. Crampton gave stovaine and ether. The only difference from the ordinary technique consisted in the disarticulation at the sacro-iliac synchondrosis without preliminary division of the ala of the ilium; such a disarticulation involves a good deal of force applied to the pelvic arch, whereby the antero-inferior aspect of the articulation is made to gape. The operation was followed by a moderate amount of shock; but from six to twelve hours after his operation his condition steadily improved, and all anxiety seemed over. Then came a period of secondary shock which defied every therapeutic measure, and death took place twenty hours after operation.

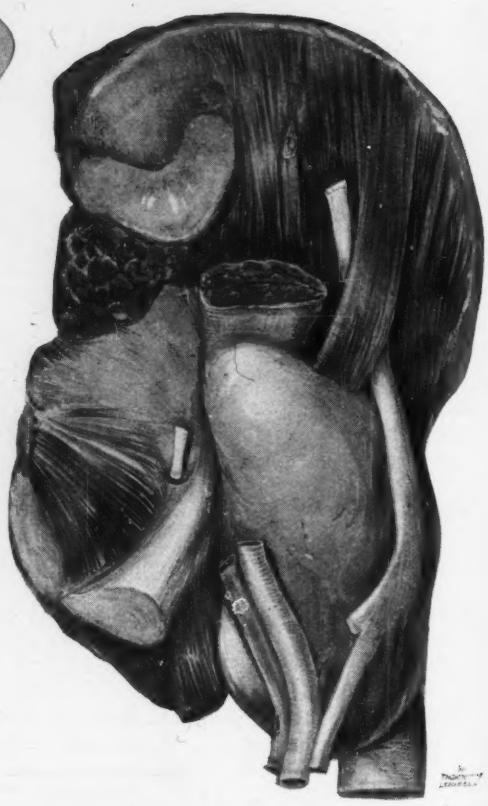

FIG. 463.—*Case* 10. Spindle-cell sarcoma of psoas muscle; tumour prominent above and below Poupart's ligament. Death from operation.

At the post-mortem there were no signs of any secondary growth present. (*Fig.* 463.)

Case 11.—**Chondrosarcoma of the left innominate bone: operation: recovery.**

Alfred T., 26 years of age, was admitted into Middlesex Hospital under my care for a rapidly growing tumour of the left buttock which had only been present for about six weeks, and which was said to be rapidly increasing in size (*Fig.* 464). Radiography revealed the presence of a large chondroma which in parts strongly suggested malignancy. There was no clinical or radiological evidence of any thoracic metastases.

A hindquarter operation was decided upon.

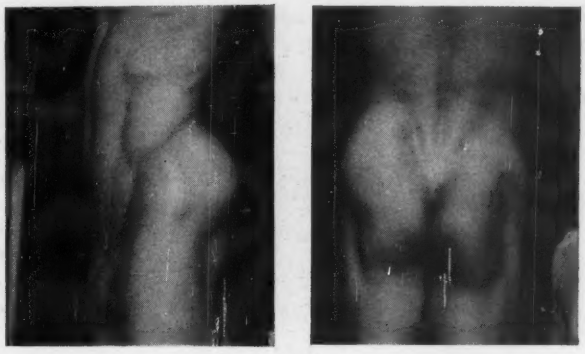

FIG. 464.—*Case* 11. Chondrosarcoma of left innominate bone. A, Viewed from side. B, Viewed from behind.

OPERATION (August 19, 1939).—Dr. Crampton gave stovaine, followed by ether. Mr. Anthony Till and Mr. F. A. Henley helped me. The transfusion arrangements were in the hands of Mr. D. D. Muir. The position of the tumour, which extended far back on the ala of the innominate bone, precluded the formation of almost any posterior flap; an attempt made to fashion an adequate anterior flap on the lines of the Savariaud technique did not succeed in covering completely the large wound. It was impossible in this case to divide the ilium from crest to sciatic notch, and disarticulation of the sacro-iliac

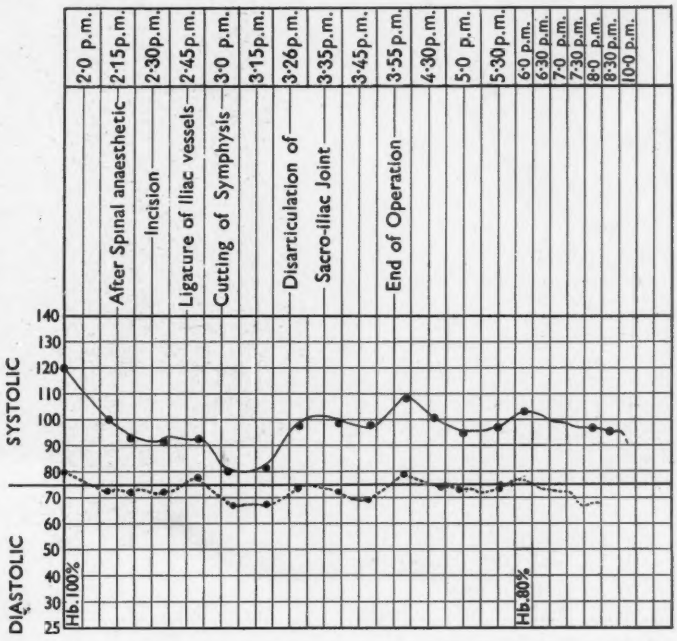

FIG. 465.—*Case* 11. Blood-pressure tracing during the operation.

synchondrosis was practised from the pelvic aspect of the articulation. The femoral artery was tied lower down than usual, and the internal iliac vessels were ligated inside the pelvis. The nerves were perhaps more liberally infiltrated with novocain than usual.

A blood-pressure tracing taken during the operation and the first succeeding hours is appended (*Fig.* 465). It is interesting that the systolic blood-pressure at the end of the

operation was only 10 mm. lower than the patient's normal, although it sank slightly in the ensuing hours. The division of the symphysis did appear to be associated with a definite fall of blood-pressure, but the forcible disarticulation of the sacro-iliac joint in this case was not marked by any depression.

The patient made an eventful recovery; his environment in hospital at the time of a political crisis was not calculated to promote the easiest convalescence, but the delayed convalescence was due to sloughing of the posterior flap.

Figs. 466, 467, show the specimen after dissection.

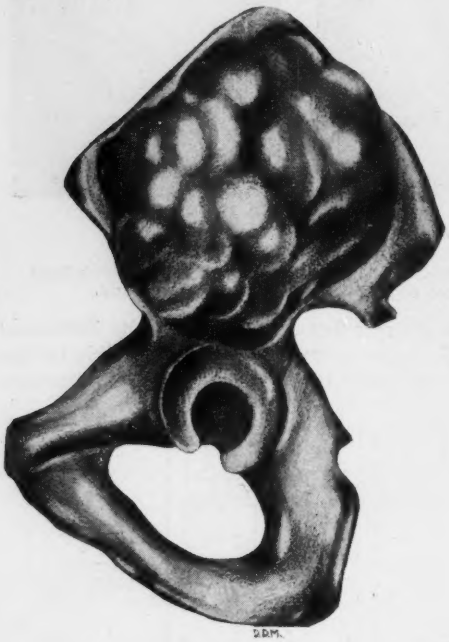

FIG. 466.—*Case* 11. Chondrosarcoma of iliac portion of innominate bone, extending to the acetabulum. Specimen after dissection; the growth had infiltrated the gluteal muscles extensively. Recovery from operation.

FIG. 467.—*Case* 11. Coloured drawing of the specimen.

DISCUSSION

Technique.—In matters of technique J. Assali and H. Sohier[2] have recently drawn attention to the advantages of a large gluteal flap in the interinnomino-abdominal operation. No description is given by these authors of actual operations performed upon their own patients, although N. Rapaut,[3] of Prague, mentions 2 cases of osteosarcoma of the femur in which he has utilized the technique of the two French writers.

It is manifestly impossible to employ this technique in cases of malignant disease involving the dorsal half of the innominate bone or the muscles of the buttock.

A good deal of attention is devoted in their paper to the conservation of the internal pubic vessels and nerves, and an elaborate manœuvre is suggested to attain this end—a truly Gallic touch!

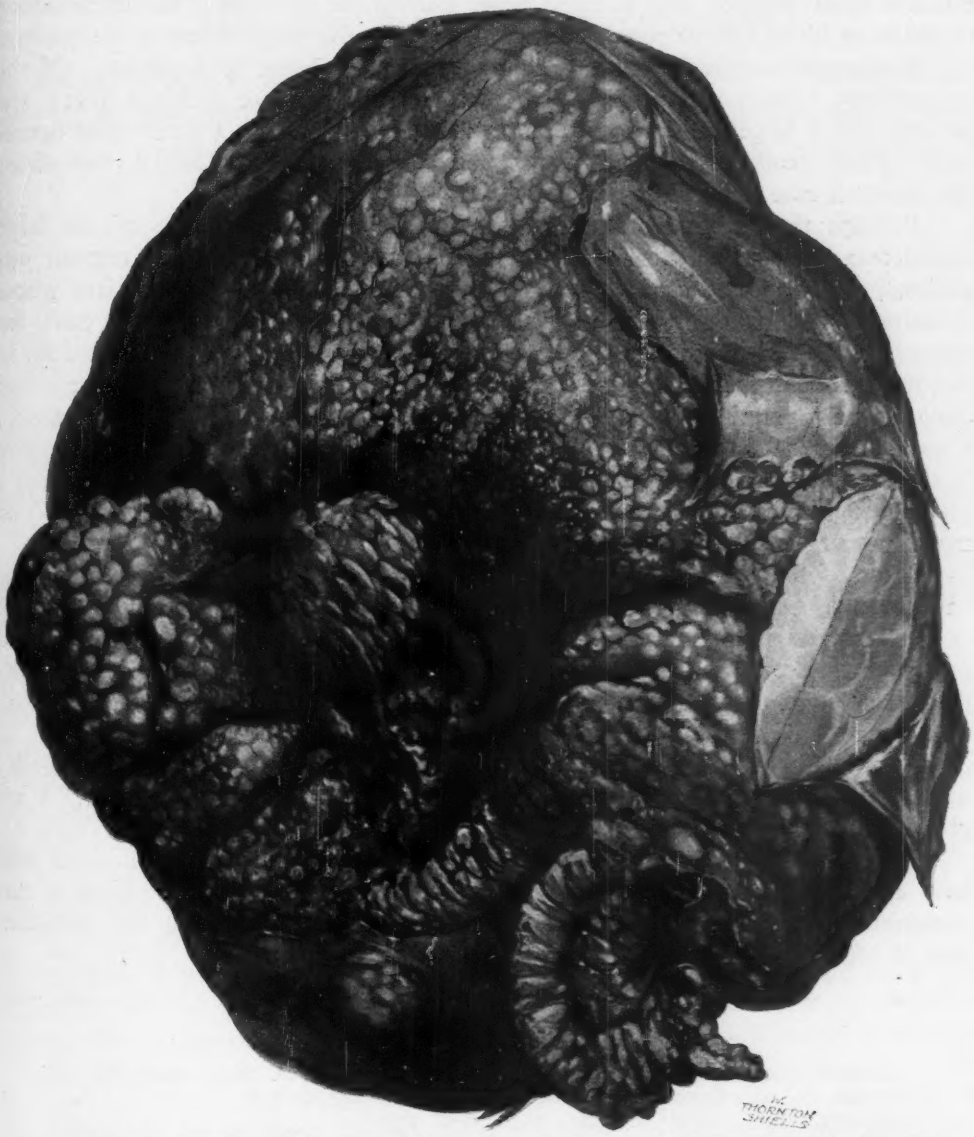

FIG. 468.—Chondroma which weighed 20 lb. and measured 20 in. in diameter. Ill-judged removal with ilium only, instead of hindquarter operation; recurrence and pulmonary metastases soon after operation.

Assali and Sohier lay stress on the desirability of bone-section rather than disarticulation of the bone; this suggestion may be rendered impossible of execution by reason of the topographical relations of the tumour; and the effect on the blood-pressure, although evident in the case of division of the symphysis, has not been discernible in the case of the sacro-iliac joint. The operation which is

suggested by the French writers, although perhaps of value in certain growths, has a limited scope.

I again affirm the desirability of nerve-blocking, both by spinal anæsthesia as well as local infiltration of the individual nerves before their division. It is wiser to institute some form of drip infusion *before starting the operation*; the introduction of saline or blood can be regulated at will, to meet the requirement of the patient.

End-results.—Death ensued from operation in 4 out of 11 cases. Of the 7 survivors, 1 case, and probably 2, developed a recurrence within a year; the patient with a large infiltrating chondroma died of recurrence 5 years after operation. Three remain free from their malady 11 years, 5 years, and 3 years after; the eleventh case is a recent recovery from operation.

Perhaps the greatest disappointment has been the recurrence of the large chondroma (*Case* 3) and its metastasis; at the time of operation the tumour was colossal, disabling, and caused great pain. A similar fate befell the patient whose chondroma is depicted in *Fig.* 468; this neoplasm had been allowed to grow for twenty years until it finally assumed a hurricane rate of growth; it was 20 lb. in weight and measured 20 in. in diameter. Neglect of those in medical charge had unconsciously permitted it to become a second largest chondroma in literature! I was most anxious to perform a hindquarter amputation, and I still regret yielding to the persuasion of others, and practising a local removal, for the girl quickly had a recurrence locally and in her lungs, proving its malignant degeneration. The major procedure of a hindquarter amputation might have preserved her life.

Most of the cases in this series have either been relatively radioresistant or have been of a size to make me doubt the permanent success of any manœuvre except the most heroic ablation. Some success of some duration has been gained by the enterprise; the happiness and the courage of my patients convince me that the preservation of life has been worth the price that they have paid in sacrifice.

Most of the illustrations have been done by my friend Mr. Thornton Shiells; while *Fig.* 466 has been drawn by my house-surgeon, Mr. D. D. Muir. I am grateful to both for their contribution to this paper.

I am under a great debt of obligation to the many friends and colleagues who have aided me in the surgical treatment of the cases which form the basis of this communication; their advice and their co-operation have played the dominant part in securing the successes that have been gained.

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A NEW CONCEPTION OF PARATHYROID FUNCTION AND ITS CLINICAL APPLICATION

A PRELIMINARY REPORT ON THE RESULTS OF TREATMENT OF GENERALIZED FIBROCYSTIC AND ALLIED BONE DISEASES AND OF RHEUMATOID ARTHRITIS BY ALUMINIUM ACETATE * †

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INTRODUCTION

THE action of the parathyroids on calcium metabolism is still not clearly understood. In 1909 MacCallum and Voegtlin[1] first demonstrated the fall in blood-calcium which usually follows parathyroidectomy. Since that time and as a result of many subsequent experimental studies it has been generally accepted that the primary function of these glands is the control of the level of calcium in the blood. Based on this premiss, many explanations of the mechanism of calcification and decalcification have been made. These theories do not always fit in with the biochemical changes obtained both clinically and experimentally—but it is the theory, and not the premiss, which has been questioned.

A few authorities, among them Pemberton,[2] Greenwald,[3] Albright,[4] Hunter,[5] and Shelling,[6] have suggested that, contrary to the above view, it is possible that parathormone exerts its primary effect on the phosphorus metabolism; but they have not applied the possibilities of this idea. It is the object of this paper to elaborate this contention and to show that treatment should have as its first object the control of phosphorus intake.

THEORY

The theory suggested is as follows:—

1. That parathormone controls the blood-inorganic-phosphate level.

2. That it prevents this level from rising to a degree which would upset any metabolic process in which the phosphates are concerned.

3. That, conversely, an accumulation of phosphate in the blood is a stimulus to increased secretion of parathormone.

4. That parathormone effects its control: (*a*) By stimulating excretion of phosphate by the kidney; (*b*) By mobilizing calcium ions from the storehouse in the bones. Following this mobilization calcium phosphate is formed. This is inactive, can be kept in solution in the blood by parathormone in greater quantities than would normally be the case, and is in a form in which it can be excreted.

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* Based on an M.D. thesis to the University of Liverpool, December, 1938, and a Report to the Association of Surgeons, May, 1939. Work done as Moynihan Fellow and as Ridgeway Research Scholar at first from the Departments of Surgical Research and of Orthopædics of the University of Liverpool and later from St. Thomas's Hospital.

† Received for publication, May, 1939.

5. That, vice versa, if the parathyroid glands are removed the body will be unable to deal adequately with the blood-phosphate. Excretion of phosphate will be diminished. Calcium ions cannot be mobilized to combine with the phosphate, and so render it inactive and excretable. The only available supply of calcium ions is the diffusible fraction of the serum calcium. J. H. Jones[7] suggested that this falls as it combines with the excess phosphate and so tetany occurs.

Basis of the Theory.—It must be remembered that the phosphate ion is an active radical. It plays a part in calcium and phosphorus metabolism, in carbohydrate metabolism and muscle activity, and is a buffer in maintaining acid-base equilibrium. An upset of its role in one of these processes may have far-reaching effects on the others. Such an upset might occur if there were any tendency for the over-production or accumulation of phosphate ions beyond the amount normally present in the blood. This excess phosphate involves the presence of an equivalent amount of basic ions both in the blood and for the purpose of excretion.

Marriott and Howland,[8] as a result of experiment, maintained that phosphoric acid, unlike other acids, does not stimulate the production of ammonia by the kidneys. The same condition obtains in the acidosis of nephritis where increased ammonia is not formed, although excess phosphate is present. In the absence of ammonia production it is essential that some other source of base be found to avoid loss of the alkali reserve of the blood. The secretion of parathormone would provide calcium for this purpose from the bones.

This interpretation fits in with experimental findings. The first effect of the injection of parathormone is an increased phosphate content in the urine, increased calcium content occurring only after an interval. After parathyroidectomy the converse takes place; elimination of phosphate by the urine is diminished and retention of phosphate occurs.

Hyperplasia of the parathyroids has been produced in rabbits by a diet excessive in phosphates and by injection of buffered phosphate solutions over varying periods.[9] Similarly many cases have been described where the retention of phosphate due to renal disease has been followed by hyperplasia of the parathyroids, osteoporosis, and the other manifestations of the syndrome of hyperparathyroidism. It is perhaps significant that Shelling and Remsen[46] have reported a case in a boy of 17 which was transitional between renal rickets and the osteitis fibrosa of adults. Moreover, it has been demonstrated that the weights of the parathyroids in a variety of chronic renal disorders exceed the normal.[10, 44, 45]

The issue is obscured by the fact that long-continued hyperparathyroidism itself causes a progressive renal failure[47]; but this may well be the explanation of the rise in the blood-phosphate level which occurs in the late stages of 'primary hyperparathyroidism', and perhaps also of the extra-osseous deposits of calcium phosphate in that condition. After prolonged administration of parathormone the blood-phosphate, which was at first reduced in level, rises, i.e., when excretion becomes difficult phosphate is retained. Calcium phosphate is formed and the blood-stream must rid itself of this burden. The extra-osseous deposits compare on chemical analysis almost exactly with $Ca_3(PO_4)_2$.[11]

In order that our theory may be maintained it must be shown that parathormone mobilizes the calcium carbonate rather than the calcium phosphate fraction of the bone-salts. We have indirect evidence in support of this. Estimation

of the relative amounts of calcium phosphate and calcium carbonate in the skeleton vary, but it is probable that the minimum proportion of calcium carbonate present is 20 per cent. That it is the calcium carbonate which is utilized is supported firstly by the deductions of J. H. Jones.[7] He prevented tetany after parathyroidectomy in rats by limiting phosphate intake. He assumed that the parathyroprival rat was unable adequately to mobilize calcium from the bones and that therefore the calcium of the blood was the sole supply available for union with the phosphate; in this manner the level of the blood-calcium fell and tetany ensued. His deduction implies that the function of parathormone is to reduce the phosphate by mobilizing the calcium ions. If calcium phosphate were mobilized the levels of calcium and phosphate in the blood would rise proportionately, whereas when the calcium alone is increased it must be that calcium carbonate is mobilized.

Further presumptive evidence is forthcoming from the work of Brehme and Gyorgy,[12] who showed that parathormone changes the *p*H of the blood without altering the carbon-dioxide-combining power. This could occur only if the change concerned the carbonate radical.

HYPOPARATHYROIDISM

If this concept of hyperparathyroidism is satisfactory the basic principles should be equally applicable to the parathyroprival state and should explain tetany.

Tetany has been produced by injecting an excess of phosphates.[13] If the dosage is gradual the parathyroids compensate, mobilize calcium from the bones, and produce fibrocystic disease; if it is rapid they are unable to deal with the excess and tetany occurs. In parathyroid tetany, blood-phosphorus is always high,[14, 15] and there is a decreased phosphorus excretion in the urine.[4, 17] Tetany has been prevented after parathyroidectomy in rats by limiting phosphate intake,[7] and the symptoms have been ameliorated by a low phosphate diet.[6, 16] It has often been observed that improvement in the symptoms of tetany is coincident with a fall in the serum-phosphorus and that a subsequent rise in the level is accompanied by an exacerbation of the symptoms, sometimes without a change in the serum-calcium content.[17]

Our hypothesis also affords the only feasible explanation of the temporary relief of parathyroid tetany by the administration of lactose or glucose.[17, 18] Feeding these sugars to patients alleviated symptoms and diminished the serum-phosphorus but did not alter the calcium level. There was no increase in the urinary phosphate. After a while the serum phosphate again rose and the symptoms reappeared. There is a relationship between carbohydrate and phosphorus metabolism. Ingestion of glucose produces a fall in serum-phosphorus for 1½ hours. Injection of insulin causes a similar reaction. This must be due to the removal of phosphate in the formation of hexose phosphate, an intermediary step in carbohydrate metabolism. So for a time phosphate ions are removed from the circulation, but as soon as the hexose has been used the phosphate ions are again liberated and the level in the blood is restored.

It is difficult to determine whether the low serum-calcium or the high serum-phosphorus is responsible for the actual manifestations of tetany. The phosphate hypothesis would permit of easier correlation with infantile tetany, many cases of which have a raised serum-phosphorus without any significant change in the calcium.[19, 20] The rise of serum-phosphate is as constant after parathyroidectomy

as is the fall in serum-calcium. On the other hand, there are the following points to consider :—

1. The onset of post-operative tetany is more frequently associated with a specific serum-calcium level of 6–7 mg. per cent than a definite phosphorus level. Brown,[21] however, reported a case in which after parathyroidectomy a patient had tetany with a blood-calcium of 9 mg. per cent, and Boyd, Milgram, and Stearns[22] have reported one in which no tetany occurred, although the blood-calcium fell to 5 mg. per cent.

2. A high-calcium diet as well as a low-phosphorus intake may alleviate the symptoms.[6]

3. In physiological experiments calcium is known to exert an effect on muscle contractility.

4. Renal dysfunction frequently produces a blood-phosphate as high as 8–11 mg. per cent, without active or even latent tetany.

The onset of tetany, however, may be prevented in this case by the compensatory hypertrophy of the parathyroids.

It is possible, of course, that the changes in serum-calcium and serum-phosphorus are but incidental processes in the production of spasmophilia and that the crucial factor is some change in the acid-base equilibrium. Hyperparathyroidism shifts the *p*H of the blood to the acid side,[12] while the induction of an alkalæmia produces tetany.

But whether the disturbance of acid-base equilibrium or of calcium-phosphorus metabolism is at fault in producing the actual syndrome, we must conclude that in post-operative tetany the chief metabolic disturbance is an accumulation of phosphate. By limiting the intake of phosphorus this can be prevented, and the value of treatment may be increased by the simultaneous administration of an assimilable form of calcium, realizing that calcium by itself does not always alleviate tetany.

PHYSIOLOGICAL (COMPENSATORY) AND PATHOLOGICAL HYPERPARATHYROIDISM

Assuming that retention of phosphate is the stimulus to increased secretion of parathormone, this ' compensatory ' hyperparathyroidism must not be confused with the classical type associated with an adenoma. In the first case, the *production is controlled by the strength of the stimulus, and contributes only that amount of hormone necessary to keep the blood-picture normal.* Clinically this is expressed by any or all of the manifestations of the syndrome ' hyperparathyroidism ', including decalcification of the skeleton and a negative calcium balance—but with a blood-calcium and blood-phosphorus within normal limits. Progress is usually slow and general symptoms not severe. (*See Cases* 2, 3, and 4.) The parathyroid glands themselves in these cases would be normal in size and appearance ; or if the stimulus were of sufficient degree, and of prolonged duration, hyperplasia of all parathyroid tissue might be expected.

On the other hand, the activity of a hypersecreting adenoma is not just the response to the phosphate stimulus. *The secretion is uncontrolled, and in excess of the needs of the body metabolism.* Blood-phosphate is reduced below the normal level by the increased excretion of phosphate in the urine. But the parallel process of mobilization of calcium is occurring, and as there are now insufficient phosphate ions to combine with it, it accumulates in the blood ; hence the high blood-calcium

and low blood-phosphorus. In Mr. Wainwright's case at the Cripples' Hospital, Stoke (not as yet reported), a blood-calcium as high as 29 mg. per cent was found in conjunction with a very large adenoma.[48]

There are two processes by which a parathyroid gland may become pathological. An adenoma may arise spontaneously in a normal parathyroid gland; or after prolonged hyperplasia of all parathyroid tissue one or more glands may become neoplastic. It is important from the point of view of treatment to distinguish these two conditions, but it is as yet impossible to do so by clinical or biochemical means before operation. A long history may be suggestive of the secondary type, but is not conclusive. In the first or primary type operative removal of the offending adenoma will restore the normal blood-picture, the symptoms will disappear, and the skeleton recover. In the 'secondary pathological' type, while excision of the neoplastic gland will stop the uncontrolled secretion of parathormone, the original stimulus is still present, and after the operation it will revert to the 'simple compensatory' type. The level of calcium and phosphorus in the blood will approach or become normal, and the acute symptoms will be alleviated; but the calcium balance will remain negative and the process of decalcification will continue. Treatment must now be instituted as for the compensatory type. *Case* 1 of my series conforms with this group, as did Mandl's first case in 1926,[23] and no post-operative increase in density of bones is reported in 7 of 15 other cases I have found in which subsequent progress was described.

An interesting correlation emerges from another analysis. Of the reports of parathyroidectomy up to 1932 only thirteen mention both the presence or absence of post-operative tetany and subsequent radiological progress in the skeleton. The results can be tabulated as follows:—

	No. of Cases	*Post-operative Tetany*	*Latent Tetany*	*No Tetany*
Improvement in radiological appearance of bones	7	5	2	–
No improvement	6	–	2	4

This suggests that the 7 cases were of the primary type and removal of the adenoma left a temporary relative hypoparathyroidism, while in the remainder operation has removed only the complication of the original compensatory hyperparathyroidism.

CLASSIFICATION OF HYPERPARATHYROIDISM

Albright[11] and his colleagues have suggested differentiating hyperparathyroidism, as follows: (*a*) Primary hyperparathyroidism due to an adenoma; (*b*) Primary hyperparathyroidism due to an idiopathic hyperplasia; (*c*) Kidney disease causing compensatory hyperplasia.

I wish to put forward an alternative classification, one that divides the condition into three definite groups as regards aetiology, pathology, and treatment.

1. *Primary Hyperparathyroidism*—due to an adenoma and typically represented by generalized fibrocystic disease, with a raised blood-calcium and reduced blood-phosphorus.

2. *Secondary Hyperparathyroidism.*—

a. Simple, compensatory, or *physiological,* resembling a mild picture of (1), but with a normal blood-calcium and blood-phosphorus. Parathyroid tissue is normal or shows general hyperplastic changes only.

b. Pathological—where an adenoma has been superimposed on (2, *a*). Clinically similar to (1), but reverting to (2, *a*) after removal of the adenoma.

THE SYMPTOMATOLOGY OF HYPERPARATHYROIDISM

The disease itself has a wide variation in intensity. It is to be expected that the pronounced manifestations encountered in the 'primary' type with a big tumour may be but feebly imitated in a slowly progressing 'simple secondary' case.

The symptoms may be divided into three groups :—

1. General.—Hypotonia, lassitude, anorexia, weakness, and general physical and mental indifference are usually present in some degree. Occasionally gastro-intestinal symptoms such as abdominal pain, vomiting, and constipation are prominent. Coldness and bluish discoloration of the extremities have also been described. In 7 cases of fibrocystic disease, 4 of which are reported here, there were skin changes which I have not been able to find described elsewhere. All were sallow in complexion, and this sallowness was accompanied in 2 cases by patches of brown discoloration of the skin and in 4 by a mild dry eruption on the face.

2. Skeletal.—These vary from cases which may show no symptoms referable to the bones to others in which the extreme softening has led to grotesque deformities.[25] In 3 of my cases of secondary hyperparathyroidism fractures occurred early in the disease. As the porosis progressed the bones gave way by bending, and splits visible on the radiograph appeared on the outside of the curves (*Fig.* 481). Cysts causing expansion appeared in 5 of the 7 cases, but occurred most profusely and most rapidly in *Case* 1 where there had been a pathological change in the parathyroid gland, and therefore uncontrolled excessive secretion of parathormone. *Bone pain* seems to depend on the intensity of the disease and the rapidity with which the bone changes occur, but *tenderness* is more constantly elicited. Complications due to the deformities of the limbs or collapse of the vertebræ are easily recognized.

In view of the opinion held by Oppel[26] and his followers[27, 28] that rheumatoid arthritis is a form of hyperparathyroidism, it is an interesting observation that pain, stiffness, and swelling of the joints are occasionally present. Funsten[29] considers that the occurrence of arthritis is frequently responsible for the first signs of parathyroid disease.

3. Renal.—An important feature of hyperparathyroidism, especially when associated with decalcification of the skeleton, is the increased excretion of calcium and phosphorus by the kidneys. The balance between the intake and output of calcium is negative when renal plus bowel output is measured against the calcium ingested. Besides, the proportion excreted in the urine as compared with the fæces is greater than the normal, i.e., to some extent excretion is diverted from the bowel to the urine. When the urinary excretion of calcium phosphate reaches or exceeds four times the normal, the diuretic effect of the increased salt concentration becomes marked and polydipsia and polyuria distress the patient. This symptom is not common in the simple secondary type.

Renal calculi, however, are a frequent finding. Wilder and Howell,[30] analysing the first 135 cases reported, ascertained that renal colic preceded evidence of disturbed calcium metabolism in 18. Albright, Churchill, Cope, and Chute as a routine measure investigate all cases of renal calculi admitted to the Massachusetts

General Hospital for evidence of hyperparathyroidism and find this disorder present without bone changes in a percentage of cases. Stasis is a contributory factor and calculi have been produced experimentally by injection of parathormone and intermittent blocking of the ureter.

Prolonged hyperparathyroidism constantly results in renal failure, usually accompanied by calcification in the kidney, and this failure is the most lethal of the complications.

THE ROLE OF HYPERPARATHYROIDISM IN VARIOUS CONDITIONS

Generalized Fibrocystic Disease.—It is no longer surmise, but can be stated definitely that any condition of decalcification of bone with or without cysts, but accompanied by a negative calcium balance, hypercalcæmia, and hypophosphatæmia, is due to hyperparathyroidism. Two conditions which fulfil these criteria we have classified as 'primary' and as 'secondary pathological' hyperparathyroidism.

The difficulty and the argument arise, however, in 'simple secondary' cases, i.e., where the blood-phosphorus and blood-calcium are normal. These have been variously named and classified as 'generalized osteitis fibrosa cystica not due to hyperparathyroidism' and as 'diffuse fibrosis of bone' by Elmslie,[32] as 'osteitis fibrosa in multiple foci' by Hunter and Turnbull,[33] and as 'osteomalacia' or 'fibrocystic disease of unknown origin' by other writers. Elmslie, and Hunter and Turnbull each had the parathyroids of one of their cases explored and no tumour was found. In the one case (*Case* 4) where we have done a calcium balance this proved to be negative before treatment was begun, although the levels of calcium and phosphorus in the blood were normal. Fairbank's case[24] already quoted is another example. It is evident that isolated estimations of the level of calcium and phosphorus in the blood give no indication of the state of the calcium balance, and it is highly improbable that a condition of osteoporosis could develop in the absence of a negative calcium balance. After a course of treatment in *Case* 4, it was found that coincident with increased radiographic density of the bones the calcium balance became positive, i.e., retention of calcium occurred.

The whole picture of the disease is similar to that of primary generalized fibrocystic disease, except that it progresses very much more slowly. There is the same sallow complexion, the atonia, the apathy, and lack of energy. These symptoms are the more noticeable when they clear up as the result of treatment, as happened in all my patients. There is a similar process of increasing osteoporosis with cysts which sometimes expand the bones. In one patient there was extensive disease of one femur and yet it was difficult at first to find definite radiographic evidence of the condition elsewhere in the skeleton. Later, involvement of both tibiæ was obvious.

All the manifestations of this form of the disease are so similar to those produced experimentally and clinically by an excess of parathormone that it is unreasonable to invoke any other aetiological factor to explain them. On reference to the case reports it will be seen that by reducing the absorption of phosphorus undoubted improvement has been produced in this condition (*Cases* 2, 3, 4). I consider that this indicates that the phosphorus radical is responsible for the excessive stimulation of the parathyroids.

Why the body should be unable to deal with the phosphates without soliciting the aid of the parathyroids, I have as yet been unable to determine. There is no obvious kidney disease, and treatment has not significantly affected blood-calcium, blood-phosphorus, *p*H, or carbon-dioxide-combining power. We have no means of judging whether a slight abnormality of the kidneys could affect the phosphate radical above all others.

Rheumatoid Arthritis.—Here, also, there is no definite evidence that a hyperparathyroid state exists. Oppel,[26] the pioneer in this field, claimed improvement in ankylosing polyarthritis after partial parathyroidectomy. Schkurov[27] obtained improvement in approximately two-thirds of his patients after parathyroidectomy, and this measure is being more commonly practised in America and in France in resistant cases of rheumatoid arthritis. There is very little biochemical support for the contention that the parathyroid does play a critical role. Oppel[26] found a raised serum-calcium in some cases, but this has not been the experience of other investigators. McCrudden[34] found a negative calcium balance in two cases of rheumatoid arthritis. A significant finding is that of Ellis,[35] who pointed out that in rheumatoid arthritis phosphoric acid is frequently absent from the urine. McCrudden[34] also found it reduced. There is some similarity to hyperparathyroidism in the clinical picture. There is the same sallow complexion, atonia, and anorexia. There is often a loss in weight. The bone ends are usually decalcified; but it may be that this is due to the immobility and is similar to the change which occurs when a limb is splinted. Leriche,[28] however, considers that the decalcification is the primary change and that the spasm and stiffness of the joints are due to the local increase in calcium affecting the ligaments and muscles. There is often a complaint of numbness and pallor of the fingers and toes in cold weather. It is possible that this is associated with a disordered calcium metabolism, and the same symptoms occur in hyperparathyroidism. Pain and stiffness of joints is often a feature, and is occasionally the first manifestation of generalized fibrocystic disease.

Two patients present features of possible significance. *Case* 8, a child 8 years of age, has a five years' history of progressive rheumatoid arthritis. When seen she was almost completely ankylosed. Radiographs showed that associated with the joint changes there were bone changes not dissimilar to fibrocystic disease. Advanced osteoporosis, considerable deformity of the bone ends, and what may be considered to be early cystic changes in the metaphysis were present. Another case, in which I have just started treatment and is therefore not reported here, had a temporary dysuria and passed a calcium phosphate stone. Eight months later he developed the typical changes of rheumatoid arthritis. The stone may have been the sign of an increased excretion of calcium phosphate by the kidney.

There is no denying the efficacy in some cases of gold therapy or of removal of a source of sepsis, but it is possible that rheumatoid arthritis has more than one ultimate aetiology and yet that the common mechanism employed is secondary hyperparathyroidism. In view of these considerations and on empirical grounds I have treated 12 cases of rheumatoid arthritis and 3 cases of spondylitis ankylopoietica with aluminium acetate. Some of these patients had previously received gold therapy, some had had a focus of sepsis removed, in all cases without effect, and the remainder were untreated. The results have been very encouraging.

Osteitis Deformans.—In 1925 Knaggs[36] attempted to correlate osteitis fibrosa and osteitis deformans. He assumed a continuous toxic basis. If the vitality of the patient is sufficient to resist the toxin until old age, Paget's disease occurs; if the reaction is only fair, osteitis fibrosa results, and if there is no reaction at all osteomalacia is the result.

We are inclined to agree that these two conditions may have a similar aetiology; the causal factor being, not a toxin, but a retention of phosphates. The different reaction would be due to the inability of the older subject to respond or compensate as adequately as the younger. By analogy, toxic hyperthyroidism manifests itself as exophthalmic goitre in the young and as 'secondary hyperthyroidism' a few decades later. An abnormal pituitary results in gigantism or acromegaly according to age. Similarly there may be three stages of 'simple secondary' hyperparathyroidism—adolescent rickets (not associated with renal damage) in the schoolboy, osteitis fibrosa in the young adult, and Paget's disease in the fifth and later decades.

Ballin considers that the results of parathyroidectomy in Paget's disease confirm the opinion that the disease is identical with osteitis fibrosa.[37] French practice is inclined to explore the parathyroids in Paget's disease.[38] On the other hand, Van Hazel and Andrews, after metabolic studies of the disease, concluded that a state of hypoparathyroidism exists. There is a retention of calcium and phosphate.[31] Bassler[39] and Colt and Lyall,[40] in agreement with this conception, have reported improvement after the administration of parathyroid extract.

In formulating these theories little consideration seems to have been paid to the fact that there are two distinct stages in Paget's disease. The first is conspicuous by marked decalcification of the affected bones. Clinically the bone is soft and deformities occur. It bends and attempts to compensate with increased bulk for its lack of rigidity. This stage is very like the hyperparathyroid state and varies in time and in degree in each case. Then follows the second stage. The affected part of the skeleton becomes radiologically of a density much greater than normal—the very opposite process. This is the time when, presumably, retention of calcium and phosphate occurs.

A more reasonable hypothesis is suggested. There is retention of phosphates. The parathyroid glands respond to their full extent, the degree to which they are able to do so controlling the degree of decalcification attained. Once they fail to compensate fully, a state of relative hypoparathyroidism exists and the reverse changes occur. This is the second and permanent stage.

TREATMENT OF HYPERPARATHYROIDISM

In view of the foregoing theoretical discussion it is reasonable that an attempt should be made in the treatment of these conditions to reduce the intake of phosphorus, and thus remove the stimulus to the over-production of parathormone.

At first an attempt was made to find an adequate diet of a constant low phosphorus content. This, however, had to be abandoned as impracticable.

An alternative method for the reduction of phosphorus intake consists in the feeding of a soluble salt of aluminium. I have found only one reference to the use of aluminium in diseases of bone, but it is of great historical interest. On Nov. 1, 1845, Dr. MacIntyre sent Dr. Bence Jones the urine of a patient suffering from 'mollities ossium'. It was in this that Bence Jones discovered the

'new substance'—'hydrated deutoxide of albumin', the urinary protein that has since been associated with his name.[41] On Nov. 15 he was asked to see the patient. "At his suggestion, alum was added to the tonics in use, with the view of checking the exhausting excretion of animal matter."[42]

Schmidt and Hoagland[43] administered an aluminium salt to four subjects and found that it was non-toxic, was not absorbed, and that it diverted phosphate excretion from the urine to the fæces. Moreover, it has also been shown that continued over-dosage with aluminium salts has produced rickets in animals.[7] Aluminium combines with phosphate in the intestine, precipitating insoluble aluminium phosphate, which is excreted unchanged by the bowel.

The acetate is the most soluble and least astringent of the salts of aluminium, and it is in this form that it has been administered. Great difficulty was experienced in finding a medium to cloak its astringency. Eventually a palatable mixture was produced by adding syrup, essence of cherry, and mel depuratum (P.B.). At Professor Drummond's suggestion I have recently started using aluminium gluconate, which is a more pleasant preparation.

No ill effects have resulted in any case from the prolonged use of small doses of aluminium acetate. Two of my patients have taken it four times a day for a year without any toxic reactions or gastro-intestinal upset. Mild and temporary constipation is occasionally experienced in the early stages, but this is readily controlled with liquid paraffin.

Since aluminium acetate is a new measure in treatment, and in view of the long course of therapy likely to be necessary, it was thought advisable to administer doses as small as possible consistent with obtaining reasonable results.

The average adult body contains about 1000 g. of calcium, 98 per cent of which is in the skeleton. A loss of calcium at the rate of 150 mg. per day for five years would amount to 28 per cent of the total skeletal calcium. In most cases the disease has been present much longer than this before treatment is started, with a correspondingly greater total loss of calcium.

An amount of aluminium acetate with a phosphate-combining power equivalent to 150 to 220 mg. of calcium was prescribed each day. Children were given a correspondingly smaller dose (the equivalent of 100 to 150 mg. of calcium per day); quarter of this daily dose was taken after each meal so that the aluminium could combine with the phosphate in all parts of the diet.

As will be seen, even these small doses produce satisfactory results, so dosage has not been varied to any great extent. It remains to be seen whether larger doses can be administered over long periods and will produce more rapid results.

The mixture I have been using was prepared for me by Messrs. Evans, Lescher, & Webb, of Liverpool, and has the following formula:—

℞ Liq. aluminium acetate (B.P.)	ʒ 1½	Ess. cherry conf. opt.	℥ 4
Syrup	℥ 360	Mel depuratum (P.B.)	ad ʒ 4

Minimum dose, ʒ 1 q.i.d. p.c.

This is combined with a minimum of one pint of milk a day.

It must be remembered that too large a dose can produce rickets.

In order to determine that it was the limitation of phosphate by itself that produced the results, no additional form of therapy was used. No calcium or vitamin D preparation was prescribed. No physiotherapy was given to any case of rheumatoid arthritis. The patients were on their normal diet. In order to

give them some source of calcium and vitamin D they took *not less than one pint of milk a day*. Some of them had been taking more than this habitually before treatment commenced.

As we have been experimenting with small doses progress has been slow, but improvement has been steadily maintained. In from four to six weeks the complexion improves and the sallowness disappears, there is a return of appetite and gain in weight, and the patient comments on his sense of well-being and increasing strength and energy. Radiological improvement in the bones is not evident before three or four months and is not marked before double that period. Possibly this could be speeded up by increased dosage, but great care must be taken to prevent over-dosage. One must remember it takes a long time before normal callus casts a radiographic shadow, and it must not be expected that rapid improvement will occur in these diseased bones. To check radiological results great care is taken to ensure that all the radiographs are taken under strictly comparable conditions.

It should be stated here that there is no basis for the exhibition of this treatment in *fractures*, *fragilitas ossium*, or *osteomalacia*.

TREATMENT OF GENERALIZED FIBROCYSTIC DISEASE

1. Primary.—This is associated with a pathological parathyroid gland which should be removed. Reducing phosphate intake would aggravate the condition and raise the serum-calcium level. After operation the patient returns to normal. It is doubtful whether aluminium acetate would accelerate recalcification, for the metabolism of phosphorus is now normal.

2. Secondary.—

a. Simple.—This is the ideal case for aluminium acetate therapy. Cases respond very well. Apparently the salt can be given over long periods. It has not yet been determined how soon it can be discontinued or whether cessation of therapy would mean retrogression of the disease. Further work might also provide a correlation between dosage and the degree of the negative calcium balance.

It is important that the bones should not be subjected to the strain of bearing weight prematurely. In *Case* 2, after an appreciable increase in density of the femur and increased strength of the muscles of the leg had been confirmed, the walking calliper of one leg was discarded. Increase in the degree of coxa vara occurred. Admittedly this is a very advanced case, but it indicates that the bones must be supported by callipers until there is no doubt that they are strong enough to bear weight. Non-weight-bearing exercises are, however, of benefit. (*See Cases* 2, 3, 4 for the results of treatment.)

b. Pathological.—The neck should be explored and the tumour removed. After parathyroidectomy the original stimulus to the disease is still present. Tetany rarely occurs (*see above*) and the calcium loss is still above normal. Aluminium acetate is then administered. Further clinical improvement follows and the skeleton recalcifies (*see Case* 1).

Treatment of Tetany.—We have had no opportunity of determining the effect of aluminium acetate on parathyroprival tetany. Its limited course after removal of a primary tumour and the comparative ease with which it clears up on giving parathormone and calcium suggest that exhibition of aluminium acetate would result in no great advantage. It may be of more use in the tetany which

occasionally follows complete parathyroidectomy, where presumably all parathyroid tissue has been removed.

Treatment of Paget's Disease.—Eight patients suffering from this disease have been subjected to the same routine of treatment as for secondary hyperparathyroidism. In each case they improved in colour and in weight. They could walk farther and do more before the onset of fatigue. Bone pain became less frequent and less intense, and in some cases disappeared. In two patients controlled radiographs suggest a slow change towards normality. There is less expansion, the outline of the cortex seems more definite, and the density of the bone is reduced. In spite of clinical improvement there is no appreciable change in the radiograph of any other patient.

A typical history is that of a man of 61 years of age who could walk short distances with the aid of a stick, but tired rapidly. His complexion was sallow and he ascribed his symptoms to his age. A routine X-ray examination during the investigation of some urinary dysfunction revealed the presence of Paget's disease. The aluminium acetate mixture was prescribed and in six months he looked healthy, had gained 10 lb. in weight, and could walk two and a half miles without fatigue.

Another patient, a minister, aged 52 years, complained of attacks of pain in his tibiæ. This was accentuated on Sundays when his duties necessitated long hours of standing. With five months of treatment the pain practically disappeared, he gained in energy and could attend to all his affairs in comfort.

Treatment of Rheumatoid Arthritis.—Although the theoretical basis for treatment by this means was not definitely established, the results have been encouraging.

Of the 15 patients, 12 improved in colour, 10 increased in weight; 9 who suffered previously from typical attacks of pain in the joints were greatly relieved; in 2 there was no improvement. The subsidence of symptoms in some joints was almost dramatic, and except for joints which were completely ankylosed when treatment commenced, movements improved. Two patients who had typical deformities of the fingers could, after four months of treatment, actively correct this. In some others the spindle-shaped swellings of the finger-joints disappeared. This occurred though no physiotherapy whatever was given. Carefully supervised and gradual physiotherapy and exercises should, however, enhance the value of this treatment.

The dose of aluminium acetate I have used has been small: 1 drachm of a 2·5 per cent solution four times a day is the average. In three or four weeks the response of the patient becomes manifest; his complexion, appetite, and energy improve and he acquires a sense of well-being. This is significant because even during remissions a patient suffering from rheumatoid arthritis complains that he never feels in really good health. It is as yet difficult to assess the value of this treatment in the different types of the disease, but I have the impression that the more chronic dry type of rheumatoid arthritis responds in a more satisfactory fashion than does the more acute wet type.

Progress has been followed clinically, biochemically, and radiologically. Clinical improvement in most cases was undoubted. Biochemically no significant change occurred in the blood-calcium, blood-phosphorus, CO_2-combining power, or blood *p*H. The blood sedimentation-rate fell in some cases. Radiologically no

obvious change was demonstrable. Charts of joint movements were compiled, and it was interesting to note that those joints which had latest become affected by the disease were the first to recover movement; but in some cases joints which had been completely ankylosed did regain some mobility. (*See Cases* 5–14.)

CASE REPORTS

Case 1.—**Secondary pathological hyperparathyroidism—generalized osteitis fibrosa cystica.**

HISTORY.—H. A., male, aged 29. Eight years pains in the back and limbs. Became round-shouldered. Gradually increasing weakness and repeated fractures of the legs. No frequency of micturition or constipation.

Radiography (February, 1936).—Generalized fibrocystic disease. Poverty of density of the bones. Erosion of the cortex and expansion. Cysts.

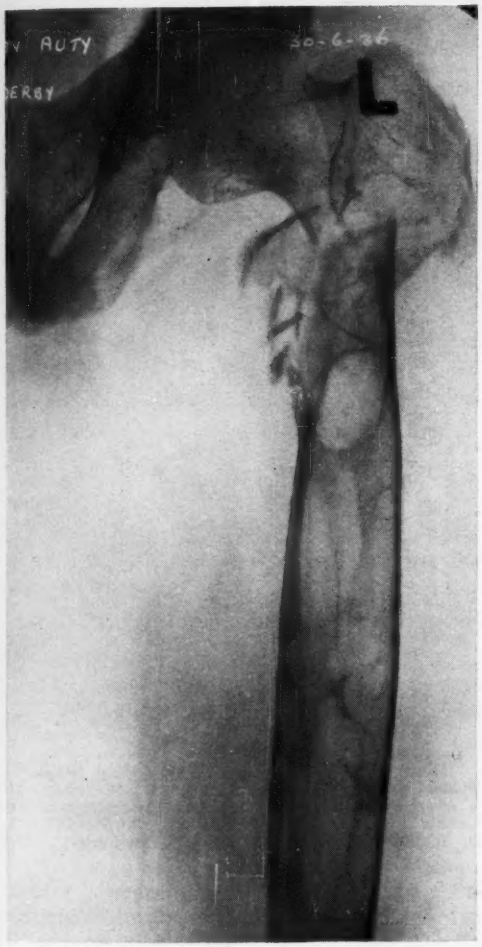

FIG. 469.—*Case* 1. Left femur, June 30, 1936, four months after removal of parathyroid tumour. Recent subtrochanteric fracture. Shaft shows advanced fibrocystic disease.

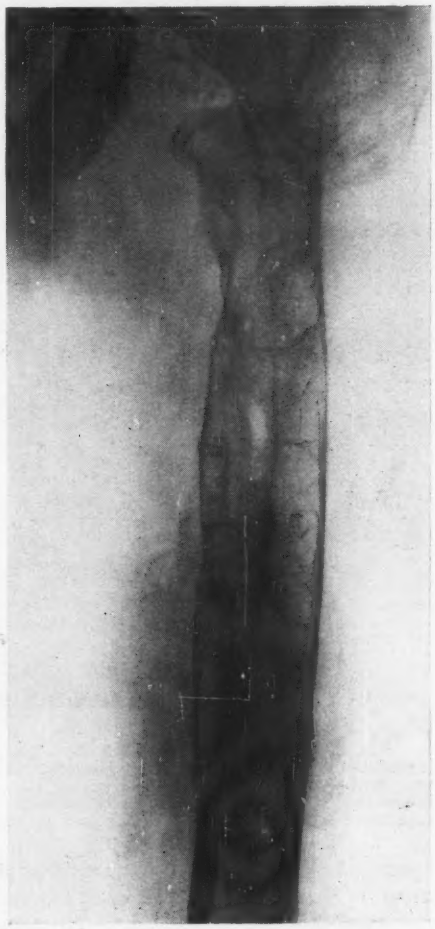

FIG. 470.—*Case* 1. Left femur, Jan. 18, 1937, eleven months after removal of parathyroid tumour. Fracture united. Changes in the shaft have markedly progressed. In particular increase in cystic change, increased expansion of shaft, and gross thinning of cortex, which now appears only as a thin pencilled line.

Calcium and Phosphorus Metabolism.—Hypercalcæmia was constantly present, the serum-calcium varying between 16·4 and 14·2 mg. per cent. The figures for plasma-phosphorus varied between 1·9 and 2·5 mg. per cent. Calcium balance determinations revealed a severe loss of calcium for each three-day period (Dr. R. H. Mole).

Blood Non-protein Nitrogen.—31 mg. per cent.

OPERATION (March 31, 1936).—Parathyroid adenoma removed by Mr. Charles Wells.

PATHOLOGICAL REPORT ON TUMOUR (Professor T. F. Hewer).—"The enlargement is due entirely to hyperplasia of the principal cells. No oxyphil cells are present. There is

FIG. 471.—*Case* 1. Left femur, Dec. 20, 1937, six months after commencement of treatment. Marked improvement in structural appearances of the bone. Cystic spaces smaller and more clearly defined. Contour more regular. Increase in thickness of cortex.

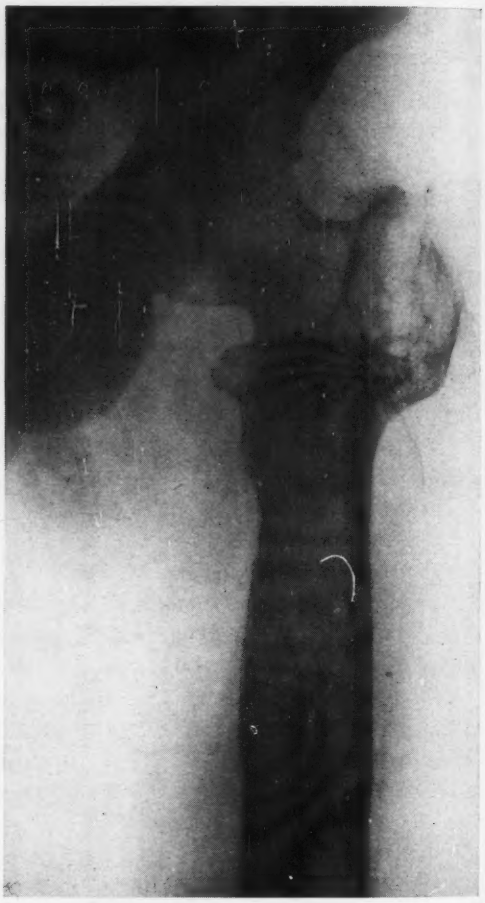

FIG. 472.—*Case* 1. Left femur, July 18, 1938, twelve months after commencement of treatment. The appearances of the bone approximate more to the normal. Pronounced recalcification. Further increase in thickness of cortex and regularity in contour.

a marked tendency to the formation of alveoli with a small amount of colloid material within them." (I am indebted to Dr. R. H. Mole for the loan of sections of the parathyroid tumour.)

POST-OPERATIVE PROGRESS.—No tetany.

Calcium and Phosphorus Metabolism.—Within 12 hours serum-calcium had fallen from 15·6 mg. per cent to 13·7 mg. per cent, and within 24 hours to 11·7 mg. per cent. Plasma-phosphorus remained constant at approximately 2·6 mg. per cent. Estimations at intervals over the next month showed that the calcium did not rise above 12·2 mg. per cent and the phosphorus remained between 2·5 and 2·6 mg. per cent. Post-operative determinations of

the calcium balance showed that this was still negative but that the amount of calcium excreted was appreciably smaller than before operation (Dr. R. H. Mole).

Calcium and vitamin D preparations were prescribed.

June 30, 1936.—Fracture upper end of shaft of left femur (*Fig.* 469).

Jan. 25, 1937.—Sound union of fracture in good position (*Fig.* 470).

Feb. 3, 1937.—Fracture of shaft of left femur at junction of distal and middle thirds.

April 14, 1937.—X-ray reports bony union. Fitted with walking calliper.

June 30, 1937.—On examination sallow-complexioned flabby male. Round-shouldered. Walks in calliper and by aid of a stick. Musculature poor. Complains that he is easily fatigued and has vague pains in back and limbs and especially right tibia. Tender expansion of mid-shaft of right tibia is palpable.

Blood-calcium 13·7 mg. per cent. Blood-phosphorus 4·1 mg. per cent. CO_2-combining power 65·6 volumes per cent.

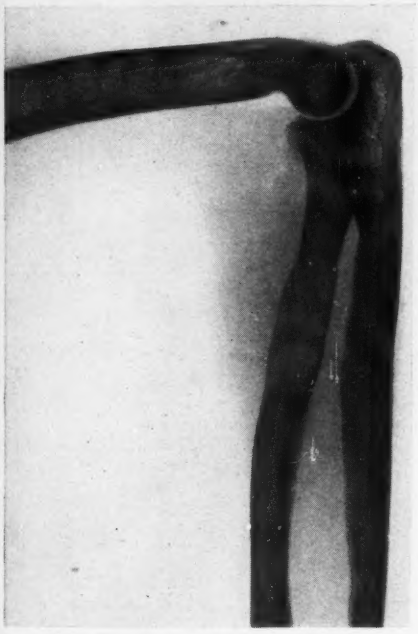

FIG. 473.—*Case* 1. Left elbow, before treatment. Large cyst in upper third of radius with expansion of shaft and thinning of cortex. Several cysts in lower end of humerus.

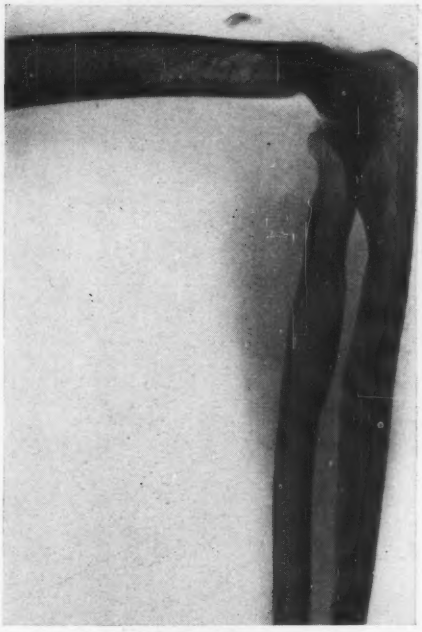

FIG. 474.—*Case* 1. Left elbow, after six months of treatment. Recalcification in radial cyst with arrest of bony expansion and increased thickness of cortex. Similar improvement in lower end of humerus.

Radiography: Generalized fibrocystic disease. Condition had become appreciably worse in the fifteen months since the operation (compare *Figs.* 470 with *Fig.* 469, and *see Figs.* 473, 475).

July 1, 1937.—Aluminium acetate and one pint of milk a day prescribed.

PROGRESS AFTER ALUMINIUM THERAPY.—Clinical, radiographic, and biochemical examinations were made at eight-week intervals.

Clinical.—Within eight weeks he had lost his sallowness. His appetite improved and he had begun to put on weight. In 12 months he gained 1 st. 9 lb. Pain disappeared. His energy and sense of well-being increased. In February, 1938, he could walk five miles along a seashore without fatigue. At first he suffered from mild constipation, but this was easily regulated by liquid paraffin, which after a few weeks could be discontinued.

Radiological.—The series of radiographs showed a steady improvement in the form and density of the skeleton. The cortex of the bones increased in depth and the girth of the cysts diminished. Radiographic density increased appreciably. (Compare *Figs.* 470, 473, 475, with *Figs.* 471, 472, 474, 476.)

Biochemical.—Every two months the serum-calcium, plasma-phosphorus, and CO_2-combining power of the plasma were estimated. Both serum-calcium and plasma-phosphorus levels fell. No change occurred in the CO_2-combining power.

On Oct. 11, 1937, that is sixteen weeks after treatment was started, the values were :—

Serum-calcium	10·8 mg. per cent
Plasma-phosphorus	0·6 mg. per cent
CO_2-combining power	65·3 vol. per cent

FIG. 475.—*Case* 1. Rght tibia, before treatment. Large cyst in middle of tibia with extreme thinning of cortex.

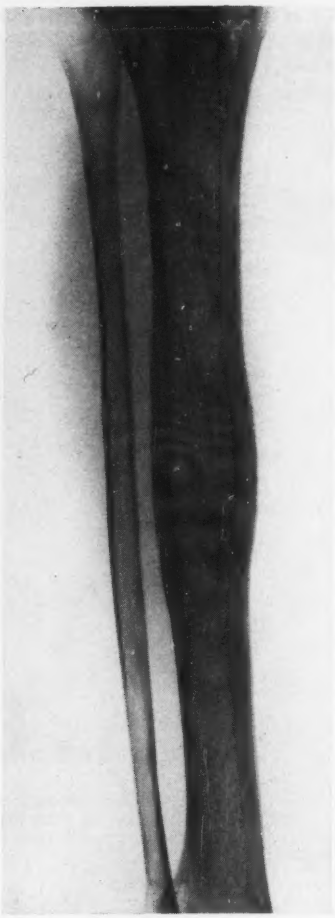

FIG. 476.—*Case* 1. Right tibia, after nine months of treatment. Marked recalcification. The thickness of the cortex now approximates to the normal. The cyst is largely obliterated.

Remarks.—Obviously removal of the tumour had not removed the cause of the disease. Subsequent administration of aluminium acetate caused rapid improvement. On July 18, 1938, this therapy was discontinued. On Sept. 9, 1938, he informed me that a few pains in his tibia had recurred. A further three months' course was given. In March, 1939, he was perfectly fit and radiographs showed further improvement.

Case 2.—**Generalized osteitis fibrosa—simple secondary hyperparathyroidism.**

HISTORY.—J. D., male, aged 17. Has at least a ten years' history of slowly progressing weakness and increasing deformity. Started with limp in left leg " because the left leg had

stopped growing". Found to be due to the development of a coxa vara on that side. He has sustained four fractures of one and three fractures of the other femur. No history of renal disease or of scarlet fever. *Fig.* 477 shows the X-ray appearance of the left femur on March 13, 1933.

OPERATIONS.—

May 15, 1936.—Oblique osteotomy of upper end of shaft of left femur by Mr. Eastwood.

July 3, 1936.—Two beef-bone pegs were inserted into the medulla of the femur above and below the site of the osteotomy by Mr. McMurray (*Fig.* 478).

POST-OPERATIVE PROGRESS.—Radiographs of the progress of the disease and fate of the bone-grafts are shown. It will be seen that in spite of the local supply of calcium and the

FIG. 477.—*Case* 2. Left femur, March 13, 1933. Advanced fibrocystic disease.

FIG. 478.—*Case* 2. Left femur, Oct. 12, 1936. Disease has progressed. There is a peculiar flattening of the outer aspect of the shaft. Osteotomy has been performed and beef-bone pegs have been inserted above and below the osteotomy.

gradual absorption of the graft, a progressive decrease in density and increase in deformity of the bone occurred. There is delayed union of the osteotomy. (*Figs.* 478, 479.)

Blood-calcium: Results of analyses varied between 11·2 and 12·5 mg. per cent.

July 19, 1937.—On examination sallow-complexioned thin youth of less than average height. Face disfigured by a dry acneform eruption. Musculature poor. Marked coxa vara and bowing of both femora. Wears walking callipers. Walking is a great effort and can be accomplished only by the aid of sticks.

Radiographs of femora, skull, and humerus show advanced generalized osteitis fibrosa. There are splits appearing at apex of curve on femur (*Figs.* 479, 481).

Blood-urea 24 mg. per cent; serum-calcium 11·5 mg. per cent; plasma-phosphorus 2·8 mg. per cent.

July 20, 1937.—Aluminium acetate and one pint of milk a day prescribed.

FIG. 479.—*Case* 2. Left femur, July 19, 1937, just before the commencement of treatment. The bone-graft is becoming absorbed; in spite of this the bone itself is becoming more porotic.

FIG. 480.—*Case* 2. Left femur, June 13, 1938, after eleven months of treatment. Increase in calcification of shaft.

FIG. 481.—*Case* 2. Right femur, July 26, 1937 Showing changes of fibrocystic disease.

FIG. 482.—*Case* 2. Right femur, March 7, 1938, after eight months of treatment. Increased calcification, most marked on outer border of shaft.

PROGRESS AFTER ALUMINIUM THERAPY.—

Clinical.—In general health and strength he showed satisfactory improvement. His complexion improved and the acne disappeared. I wish to remark on this latter change. When some time later by an error he did not get his aluminium acetate for three weeks these spots recurred. They disappeared when the mixture was again taken. After some months he was able to walk much more freely for greater distances. He stated that he felt stronger and more energetic.

Radiological.—Gradual increase in density of the bones occurred (*see Figs.* 480, 482).

Biochemical.—No significant change was demonstrated by a series of blood analyses.

Remarks.—Of the 5 cases of generalized fibrocystic disease reported here, this patient has made the slowest progress. Probably the dosage in his case should have been larger. On June 13, 1938, as the radiographs showed an appreciable increase in density of the femora, one calliper was discarded. After a few weeks the varus deformity of the neck of that femur had increased perceptibly. He was taken into the ward and traction applied. Recently his general condition deteriorated, and radiographs of the chest revealed that he has developed a bilateral phthisis.

Case 3.—**Generalized osteitis fibrosa cystica—simple secondary hyperparathyroidism.**

FIG. 483.—*Case* 3. Right femur, July 22, 1937. Fibrocystic disease. Multiple cysts. Fracture through the largest of these.

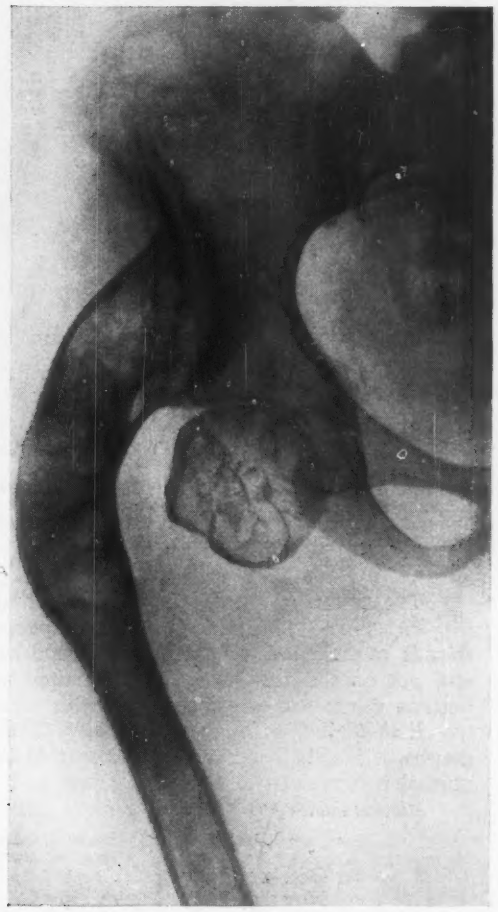

FIG. 484.—*Case* 3. Right femur, Sept. 3, 1938, after twelve months of treatment. Fracture united. Increased consolidation not only at site of fracture but in all cysts. Increase in thickness of cortex especially well shown in upper and outer end of shaft.

HISTORY.—J. M., male, aged 39. Twenty-five years' history of slowly increasing weakness and deformity. For some years he had been unable to give much attention to his business and had been in and out of hospital with fractures of the lower limbs. Deformity had reduced his stature and the development of cysts in the skull bones had given him an amazing appearance. He stated that the deformity of the thighs had been the first sign, the changes in the head and mandible occurring later. No history of renal disorder or scarlet fever. On July 21, 1937, he was admitted to Mill Road Infirmary with fracture of the shaft of the right femur.

ON EXAMINATION (Sept. 8, 1937).—Brownish-complexioned patient; looks older than his years. Cysts palpable on skull and mandible. Gross deformities of both femora. Marked coxa vara both hips.

Radiography.—Shows advanced generalized fibrocystic disease. Poor density of bones. Ununited fracture of neck of right femur; head detached and well below great trochanter. Fresh fracture through cyst in shaft of right femur. Skull shows cysts, and in parts the fuzzy outline and calcification resembling Paget's disease. (*Figs.* 483, 485.)

Biochemical.—Renal function: Results of urea-concentration tests within normal limits. Serum-calcium 8·9 mg. per cent; Plasma-phosphorus 3·1 mg. per cent; CO_2-combining power 68·1 volumes per cent.

FIG. 485.—*Case* 3. Skull, July 22, 1937, before treatment. Advanced fibrocystic disease. Note cyst-like spaces in parietal region. 'Woolliness' of outer table of frontal bone.

FIG. 486.—*Case* 3. Skull, Sept. 3, 1938, after twelve months of treatment. The cyst-like spaces have almost disappeared. There is a greater homogeneity of bone structure. Outer table of frontal bone has acquired its normal smoothness.

TREATMENT (Sept. 8, 1937).—Aluminium acetate and one pint of milk per day prescribed.

PROGRESS.—The patient improved perceptibly. After a few months he stated that his friends remarked that he had not looked so well for years. He became much more active and put on weight. The fracture united and the calliper was discarded. Occasionally he became somewhat constipated, but this was not unduly troublesome.

Radiological.—Slowly the density of the bones increased. In comparing later radiographs of his skull it may be noticed that the outline is more defined and the bone is more normal in appearance. (*Figs.* 484, 486.)

Biochemical.—

Date	*Serum Calcium mg. per cent*	*Plasma-phosphorus mg. per cent*	*CO_2-combining power vol. per cent*
Oct. 14, 1937	8·8	3·5	68·3
Nov 15, 1937	9·5	2·8	69·1
Jan. 24, 1938	9·2	2·5	76·7
Sept. 2, 1938	10·09	2·59	75·1

The serum-calcium has slowly increased in level, while the plasma-phosphorus has diminished.

Remarks.—There is undoubted improvement in this patient. It is remarkable that he walks so well in spite of the ununited fracture of the neck of the right femur. Radiographs in February, 1939, showed that his improvement continues. He is leading an active life and has had no fracture for nearly two years. I think a larger dose of aluminium acetate would have speeded his recovery.

Case 4.—**Generalized osteitis fibrosa cystica—simple secondary hyperparathyroidism.**

HISTORY.—L. T., male, aged 16. At least six years' history of increasing weakness and deformity elicited. No history of scarlet fever or renal disease.

OPERATIONS.—

June, 1936.—Osteotomy of left femur by Mr. B. L. McFarland.

October, 1936.—Osteotomy of right femur by Mr. B. L. McFarland.

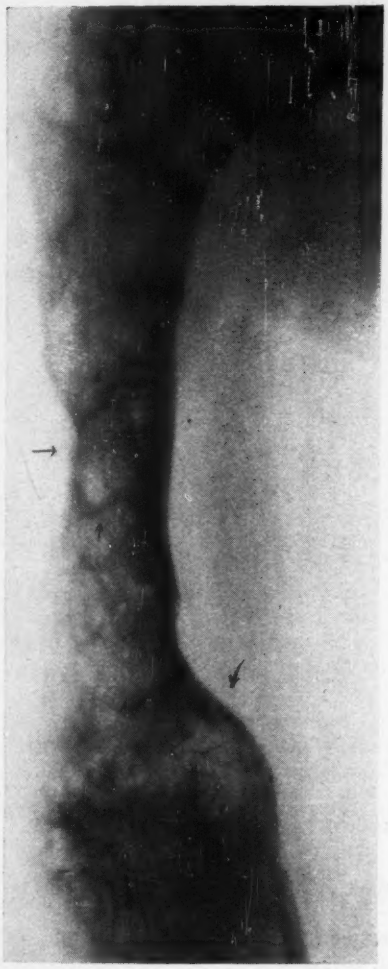

FIG. 487.—*Case* 4. Right femur, Feb. 7, 1938, before treatment. Advanced fibrocystic disease. United fracture.

FIG. 488.—*Case* 4. Right femur, Aug. 23, 1938, after six months of treatment. General increase in consolidation. Contour of bone more regular.

POST-OPERATIVE PROGRESS.—Both osteotomies united and he was allowed to walk in two walking callipers. Appearance of thighs had been much improved (*Figs.* 487, 489).

Jan. 15, 1936.—Blood-urea 28 mg. per cent; Plasma-phosphorus 1·66 mg. per cent; Serum-calcium 10·2 mg. per cent; Wassermann reaction negative.

Radiography: Fibrocystic changes in femora, tibiæ, and pelvis. Skull suggestive of Paget's disease.

March 4, 1938.—On examination sallow, tall youth with patches of brown pigmentation on the face. Musculature poor. Disinclined to exert himself. Both femora bowed. Walks with difficulty in two callipers. Head large and skull irregular. Has long, spatula-shaped fingers.

Radiography: Shows advanced fibrocystic disease. Poor density of bones. (*Figs.* 487, 489.)

FIG. 489.—*Case* 4. Left femur, Feb. 9, 1938, before treatment. Fibrocystic disease. Decalcification.

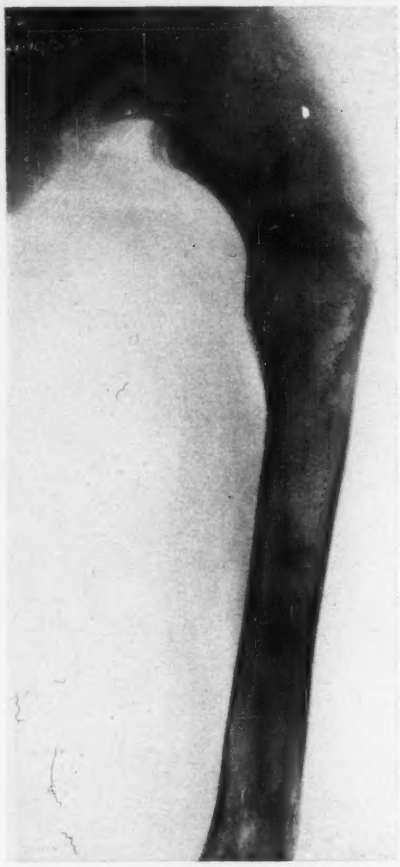

FIG. 490.—*Case* 4. Left femur, Aug. 23, 1938, after six months of treatment. Greater thickness of cortex. General recalcification.

Biochemical: Dr. Gaddie's results before and after giving aluminium acetate are reported below.

March 7, 1938.—Aluminium acetate and at least one pint of milk a day prescribed.

PROGRESS AFTER ALUMINIUM THERAPY.—Clinical, radiographical, and biochemical improvement were very satisfactory. The boy's appetite and sense of well-being increased and in six months he had put on nearly 1 st. in weight. Calcium balance experiments showed that, instead of losing, he was now storing calcium. He was walking more freely and far greater distances without fatigue. He could now stay on his feet for hours on end. He was not constipated.

Radiological: Increase in density of the bones and greater depth of the cortex occurred (compare *Figs.* 487, 489 with *Figs.* 488, 490).

Biochemical :—

Date	*Blood pH*	*Blood-calcium mg. per cent*	*Blood-phosphorus mg. per cent*
March 4, 1938	7·30	10·05	3·14
March 5, 1938	7·40	10·30	3·67
March 6, 1938	7·42	10·20	3·44
March 7, 1938, began taking aluminium acetate and milk			
April 4, 1938	7·25	11·05	3·24
April 5, 1938	7·27	10·70	3·10
April 6, 1938	7·25	10·60	3·30
May 3, 1938	7·35	10·65	3·02
May 4, 1938	7·38	10·63	3·23
May 5, 1938	7·41	10·84	3·38
Aug. 23, 1938	7·35	10·60	3·34

Calcium and Phosphorus Balance Experiments :—

March 2–6	Negative
April 2–6	Positive
May 1–5	Positive

The intake was analysed chemically as well as the output:

March

Calcium intake	3·284 g.	Phosphorus intake	3·112 g.
Calcium output	4·478 g.	Phosphorus output	3·549 g.
	− 1·194 g.		− 0·437 g.

April

Calcium intake	4·294 g.	Phosphorus intake	3·856 g.
Calcium output	2·930 g.	Phosphorus output	3·248 g.
	+ 1·364 g.		+ 0·608 g.

May

Calcium intake	2·856 g.	Phosphorus intake	3·233 g.
Calcium output	2·263 g.	Phosphorus output	3·357 g.
	+ 0·593 g.		− 0·124 g.

Remarks.—This patient, whose condition both clinically and radiologically had been steadily deteriorating for years, is now definitely improving.

Case 5.—**Rheumatoid arthritis.**

HISTORY.—E. J., female, aged 26. Eleven weeks ago perfectly well and active. Since then attacks of pain and swelling in wrists, hands, and feet. Joints very stiff in mornings. More recently occasional pains in neck and shoulders. Complains of lassitude. No abnormal renal symptoms.

ON EXAMINATION.—Very sallow complexion. Wrists, both swollen and movements restricted by one-third. Spindle-shaped swelling of the metacarpo-phalangeal and some interphalangeal joints. Feet, both swollen over dorsum. Limitation of dorsiflexion and inversion.

TREATMENT.—Aluminium acetate and one pint of milk a day prescribed.

PROGRESS.—In six months she had practically recovered. Her health had improved, colour was good, and she had gained 12 lb. in weight. There was no swelling of any joint and her only symptom was aching of the left wrist after she had been typing for some time.

Case 6.—**Rheumatoid arthritis.**

HISTORY.—F. D., female, aged 23. Twelve months' history of rheumatoid arthritis of left knee, both wrists, fingers, and ankles. Severe pain, especially of knee. Patient has been to Buxton without improvement. After an attack of influenza she developed albuminuria which disappeared. In November, 1937, she was given three injections of gold salts (myocrisin), but as the albuminuria reappeared it was discontinued. No focus of sepsis found.

ON EXAMINATION (Jan. 17, 1938).—Anæmic-looking girl. Left knee swollen. Unsound ankylosis. Limitation of movement and swelling of both wrists, ankles, and some fingers. Renal function normal. Urine: specific gravity 1016–1022; reaction acid; albumin nil.

Wassermann reaction negative. Sedimentation-rate, raised. Walking in left calliper, but limited by pain and early fatigue.

TREATMENT (Jan. 18, 1938).—Aluminium acetate and one pint of milk a day prescribed.

PROGRESS (May 22, 1938).—Has gained 10 lb. in weight and feels and sleeps well. Walks without calliper and without pain or early fatigue. Knee ankylosed. Full movements of wrists, which are hardly swollen. Is returning to work—private nursing.

Remarks.—Patient improved considerably.

Case 7.—**Rheumatoid arthritis.**

HISTORY.—W. H., female, aged 26. Four years of intermittent exacerbations of pain, swelling, and limitation of movement of the hands and feet. Occasional attacks in shoulder, back, and left knee. One course of gold injections in 1937 without effect.

ON EXAMINATION (March 2, 1938).—Sallow complexion. Slight swelling and stiffness of wrists and the metacarpo-phalangeal joints of both index fingers. Swelling on dorsum and round lower end of the Achilles tendon of both feet. Limitation of movement of ankles and toes. Non-protein nitrogen, 25 mg. per cent.

Radiographs.—Typical of rheumatoid arthritis.

TREATMENT (March 3, 1938).—Aluminium acetate and one pint of milk a day prescribed.

PROGRESS.—The general condition of the patient rapidly improved. She gained in colour and in weight. The intervals between attacks lengthened and the attacks themselves decreased in intensity and duration. From May onwards she had very little trouble. By July all swelling had disappeared from the feet, wrists, and right index finger. The base of the left index finger was still spindle-shaped, but not painful. She still has very occasional but not severe pain in wet weather. Her hands have not yet regained their full power.

Remarks.—The patient has made excellent progress.

Case 8.—**Rheumatoid arthritis.**

HISTORY.—J. E., female, aged 8 years. Five years' history of rheumatoid arthritis. Her feet were affected first, then the knees, hips, arms, and spine. Movements of the joints became progressively more limited. For two years she has had no pain, but has been completely helpless. She cannot feed herself and can just raise her head to look about. Appetite good. Previous treatment: tonsillectomy, myocrisin injections, physiotherapy.

ON EXAMINATION.—Healthy-looking child. Skin brownish in colour, but probably due to artificial sunlight. Child lies on her back and can just raise her head enough to see her feet. Back stiff and limbs practically ankylosed. Flexion deformity of both hips and knees. Both elbows ankylosed at a right angle and can bring neither hand to her mouth. Swelling of knee-joints, elbows, wrists, and fingers. Weight 35 lb.

Radiography.—Show marked atrophic changes of the bones. Parts of the femora are so translucent that they give the appearance of small cysts in the bones. The bone-ends at the knee, hip, and elbow are deformed and the radiographs suggest bony ankylosis of the knee-joints and elbow-joints.

Biochemical.—Urine, nothing abnormal. Blood-urea, 25 mg. per cent. Urea clearance (Van Slyke), 83 per cent of normal function. Serum-calcium, 10·5 mg. per cent. Plasma-phosphorus, 3·47 mg. per cent.

TREATMENT (May 27, 1938).—Aluminium acetate and a minimum of one pint of milk a day prescribed.

PROGRESS.—The improvement in this child was almost dramatic. In three months she gained 5 lb. in weight and was brighter and more active. The swelling of the joints except for her right wrist had disappeared. She could raise her shoulders from the couch, could feed herself with her right hand and undo buttons with her fingers. On Sept. 2 heat and light massage was prescribed in addition to the aluminium salt. Four weeks later she could raise part of her trunk, roll over on to her face and crawl a little. Her right hand could be brought right to her mouth, but her left hand was still two inches short of it. She had lost 1 lb. in weight in the four weeks, but apparently had perspired freely when heat was administered.

Radiological (Sept. 2, 1938).—Increased density of the shafts of the long bones.

Renal Function (Sept. 2, 1938).—Blood-urea, 30 mg. per cent. Urea clearance, 134 per cent of normal function.

Remarks.—The improvement in this case is astonishing. It is worthy of notice that the joints affected latest by the disease have been the first to improve.

Case 9.—**Rheumatoid arthritis.**

HISTORY (Jan. 24, 1938).—I. L., female, aged 8 years. For 18 months has had recurrent attacks of pain and swelling in the hands and feet with occasional involvement of the knees. Her present complaints were of swelling of the hands and feet, with some limitation of movement. Stiffness of the ankles after rest. Poor and capricious appetite.

ON EXAMINATION.—Sallow-complexioned child with pouches under her eyes. Tall for her age, but rather thin. Weight, 3 st. 11 lb. Periarticular swelling of both wrists and metacarpo-phalangeal joints of both hands. Rather less on dorsum of feet and round ankles. Tenosynovitis of extensors of left wrist and left toes.

Movements.—Right wrist—extension 40°, flexion 65°; left wrist—extension 60°, flexion 60°; ankles and feet—just short of full. Inversion difficult.

Radiography.—Slight atrophic changes of bone-ends near wrists and ankle-joints.

Biochemical.—Blood-urea, 29 mg. per cent. Urea clearance (Van Slyke), 51·35 per cent of normal function.

TREATMENT (Jan. 24, 1938).—Aluminium acetate and a minimum of one pint of milk a day as prescribed.

PROGRESS.—The child improved steadily. Within two months her colour and general appearance spoke of better health. Her mother commented on her improved appetite, her less-troubled sleep, and increased energy. She went more willingly to school. The pouches under her eyes disappeared. In six months, however, she gained only 1 lb. in weight. The swelling of the joints disappeared within three months, the effusion into the tendon-sheaths being reduced more slowly. The movement of both ankles was full and that of the wrists improved considerably. She is now in her gymnasium class at school.

Renal Function (April 25, 1938).—Blood-urea, 24 mg. per cent. Urea clearance (Van Slyke), 70·2 per cent of normal function.

July 18, 1938.—Aluminium acetate discontinued. Easton's syrup prescribed.

Feb. 19, 1939.—General health maintained. No recurrence of symptoms.

Remarks.—Child definitely improved.

Case 10.—**Rheumatoid arthritis.**

HISTORY (June 15, 1938).—T. M., male, aged 61. Eight months' pain in left thigh. No pain elsewhere. Pain varies in intensity and is relieved by rest. Various joints swell occasionally, especially knees. Appetite good, but has lost a little weight.

ON EXAMINATION.—Sallow complexion; unhealthy-looking. Swelling of left knee. Pronounced creak. Flexion short of a right angle. Urea concentration within normal limits.

TREATMENT.—Aluminium acetate and milk prescribed.

PROGRESS (Sept. 19, 1938).—Looks much healthier. Colour good. Has put on weight. No swelling of either knee. Full movement of left knee. Is working as a night-watchman.

Case 11.—**Rheumatoid arthritis—spondylitis ankylopoetica.**

HISTORY (Feb. 8, 1939).—L. S., male, aged 24. Four years of intermittent pain in left hips and lumbar spine and increasing stiffness of back.

ON EXAMINATION.—Thin sallow man. Lumbar spine rigid. Can barely touch knees with hands. Left hip, pain at extremes of movement.

Radiography.—Some calcification of the ligaments of the lumbar spine.

TREATMENT.—Aluminium acetate prescribed.

PROGRESS (Aug. 10, 1939).—Has put on 1½ st. in weight. No pain in hip or spine. Can bend to within 3 in. of his toes. Hips full movement. Is returning to light work.

Case 12.—**Rheumatoid arthritis—spondylitis ankylopoetica.**

HISTORY (Nov. 5, 1938).—E. C., male, aged 31. Eight years of increasing pain and stiffness of the back. Never feels really well. Early fatigue. Since 1935 has worn a posterior spinal support which has relieved the pain, but his back aches after exertion.

ON EXAMINATION.—Sallow, drawn face. Thin. Lumbar and dorsal spine completely rigid. A little pain on extremes of movement of one hip. Radiographs show typical calcification of ankylosing spondylitis. Aluminium acetate prescribed.

PROGRESS (Oct. 11, 1939).—Improved in health and in appearance. Has had no pain for months and can take moderate exercise without fatigue. Movement can be elicited between the spines of the vertebræ. He can reach to 5 in. below the patella.

Case 14.—**Rheumatoid arthritis.**

HISTORY.—L. B., male, aged 51. One year's history of irritable health. Recurrent aches and pains in various joints and acute pain in the shoulder and hip if he overswung at golf.

ON EXAMINATION.—Thin, sallow, and unhealthy-looking. Early rheumatoid arthritis.

TREATMENT.—Aluminium acetate prescribed.

PROGRESS.—In three months his colour had improved and his face had filled out. He had had no pains for some weeks and could play golf with ease.

CONCLUSION

It is with due regard to their immaturity that I present my results; but I trust that they are promising enough to merit the hope that this work will open up a new field of investigation, and a new line of treatment for these distressing diseases.

Acknowledgements.—I wish to thank Sir Robert Kelly, Professor T. P. McMurray, Mr. W. R. Bristow, Professor H. Channon, Professor J. C. Drummond, and Professor W. J. Dilling for the great encouragement and valuable advice and help they have given me.

I must acknowledge a great debt of gratitude to Professor T. F. Hewer and Dr. R. Gaddie, who have given me their unstinted aid and collaboration. Both, by virtue of their more scientific outlook, have prevented me from drawing any unreasonable conclusions. Between them they have done all the complicated and time-consuming biochemical analyses I report. In addition I have constantly appealed to them on pathological and biochemical problems.

It is with great pleasure that I record the ready kindness and help which I received from all the members of the teaching school and of the staffs of the various hospitals I approached in the course of this work. Dr. E. L. Rubin and Miss N. Walker took great pains in improving my radiological results. Mr. Watson Jones, Mr. Eastwood, Dr. Capon, Mr. McFarland, Mr. Roberts, Dr. Brookfield, Dr. Baker-Bates, Dr. McEwan, Dr. Crosbie, Dr. Findlay, and Mr. Armstrong have all allowed me easy access to cases. I must also thank the registrars and resident and nursing staffs of the various hospitals for their willing co-operation.

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ANNULAR PANCREAS

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Reports on annular pancreas have appeared from time to time, but on the whole it is a rare abnormality. In a recent survey of the literature McNaught was only able to trace 44 cases. The purpose of this paper is to record another case which was found during a routine post-mortem examination.

CASE REPORT

History.—A man, aged 65, had been treated for three years for hæmaturia caused by papilloma of the bladder. At first he was given diathermy, but when a growth appeared which had malignant characters he had radium applied. Subsequently he had a suprapubic cystotomy performed, but died shortly afterwards in uræmia. At a post-mortem the clinical diagnosis was confirmed. The bladder wall showed great thickening and both ureters had become compressed. On the left side a hydronephrosis and on the right a pyonephrosis was present. The only secondary growths found were in the aortic glands and in some glands in the pouch of Douglas. On examining the duodenum a band of pancreatic tissue was seen surrounding it in the second part (*Fig.* 491). At no time in his life had the patient suffered from any gastro-intestinal symptoms.

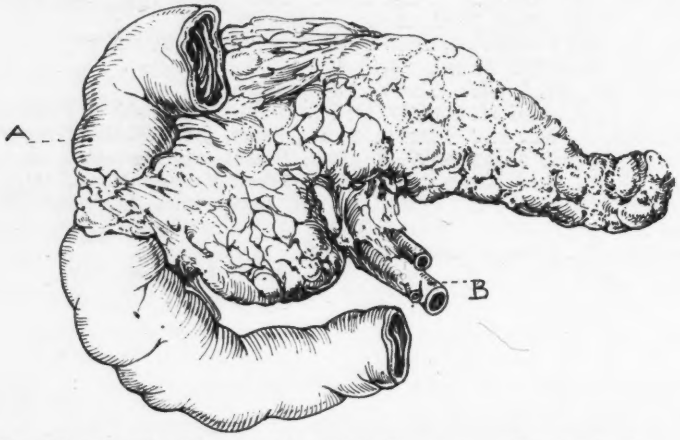

Fig. 491.—Showing the ring of pancreatic tissue surrounding the duodenum.
A, Duodenum; B, Superior mesenteric artery.

Details of the Annulus.—It appeared to consist of pancreatic tissue throughout. In some cases known as pseudo-annular pancreas the ring has been completed by fibrous tissue, but this was not so in the present case. A portion of the ring taken for histological examination confirmed the fact that it consisted of normal pancreatic tissue. Although the ring was firmly applied to the duodenum there was no marked constriction of the lumen, and the tip of the little finger was easily admitted. For the purposes of demonstration the duodenum was filled with formalin and this gave a false impression of the degree of constriction, as can be seen in the illustration. The band of pancreatic tissue was narrow throughout, but more so in its anterior part. In width its measurements were 16 mm. anteriorly, 18 mm. laterally, and 21 mm. posteriorly. It was firmly fused with the pancreas in front and behind.

Study of the Duct System.—Before any opinion as to the aetiology of the condition could be expressed a study of the duct system was essential. In order to do this and at

the same time not to spoil the specimen it was decided to adopt the procedure recommended by McNaught. A 20 per cent solution of bismuth oxychloride in 10 per cent gum acacia was used for injection and the specimen subsequently X-rayed. The injection was made through an ordinary 10-c.c. syringe and a finely pointed glass cannula used to insert into the duct. The tail of the pancreas was cut off about 1 in. from its end and the duct identified. An injection was then made and excessive pressure was avoided owing to the possibility of making false passages. Although no bismuth was seen to issue from the ampulla of Vater a subsequent radiograph showed the main duct to have filled satisfactorily. The entrance of the accessory duct into the duodenum could just be seen, but there was no sign of a duct system in the annulus. The ampulla of Vater was therefore opened up and the entrance of the main pancreatic duct into the common bile-duct found. An injection was made into the main duct, but with no improvement in the X-ray picture. The accessory duct was unchanged and that of the annulus was not in evidence. It was therefore decided to cut across the annulus on its postero-lateral aspect and to make an injection into the ends of the duct. These were easily identified and it was noted that bismuth cream issued from

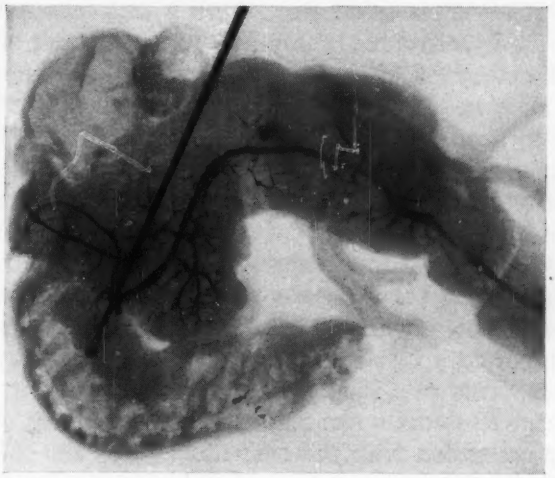

FIG. 492.—Radiograph of specimen after injection with bismuth cream. The metal probe is in the bile-duct. The main duct and the separate duct to the annulus are clearly outlined, while the accessory duct can be faintly distinguished.

the ampulla of Vater when the posterior half of the duct was injected. It was then assumed that the blind end of the duct lay in front and that the duct ran antero-posteriorly. The X-ray picture showed the annular duct to have filled satisfactorily. The posterior end ran into the common bile-duct and the only possible branch joining the main duct was an extremely small one. The latter was so doubtful that it was decided to have stereoscopic radiographs taken to clarify matters. It was then obvious that the annulus had a duct system of its own which ran into the common bile-duct on a slightly posterior plane to the entrance of the main pancreatic duct. Furthermore, the supposed connexion with the main duct lay in a totally different plane, thus proving that no such connexion existed. (*Fig.* 492.)

DEVELOPMENT OF THE PANCREAS

The pancreas develops from a ventral and a dorsal bud which lie in relation to that part of the primitive alimentary canal which will later form the duodenum. The ventral bud is bifid and by a rotation of the primitive gut comes to lie behind the dorsal bud and ultimately to fuse with it. The dorsal bud gives rise to the bulk of the organ, i.e., the tail, body, neck, and ventral part of the head. The left half of the ventral bud is said to atrophy and disappear, while the right half gives rise to the rest of the head and the uncinate process.

THEORIES OF CAUSATION OF ANNULAR PANCREAS

The theories advanced to account for this anomaly fall into two groups: (1) A simple hypertrophy of the pancreas; (2) An error of development of the pancreatic anlagen.

In support of the hypertrophic theory it has been noted that the annular pancreas has no marked abnormality of its blood-supply or arrangement of its excretory ducts; that the ring is thickest where it joins the head and thinnest at the right-hand edge, which is the point most distant from the pancreas proper; lastly that pseudo-annular pancreas is probably an early or arrested stage of the true annular pancreas, being incomplete because of insufficient hypertrophy. Villar and Lerat have slightly modified this view by suggesting that the annulus is a regenerative lobe following a fœtal pancreatitis which had subsided. Weissberg also leans toward the hypertrophic theory in a recent account of a case. He made the remarkable discovery of an annular pancreas in an embryo 16 mm. long. Unfortunately he does not give any details as to his method of studying the case and it is difficult to form any opinion on it. In the present case it would seem impossible to accept the theory of hypertrophy. In the first place the ring is narrowest in front and widest behind, and there is no central narrow part (i.e., at the right-hand border) such as is emphasized by Weissberg. Secondly the annular duct ends blindly anteriorly, thus suggesting its derivation from one rudiment and not from a hypertrophy of two parts of the pancreas. Again, were the theory of hypertrophy correct the ring should be incomplete laterally, but this is not usually the case. Thür reported a case in which a gap in the ring was filled posteriorly with fibrous tissue. This case is very unusual, however, as it is the only one on record where the annular duct opens into the accessory duct, although it had also a small subsidiary connexion with the main duct.

The majority of writers favour the embryological theory, and the ventral bud is the rudiment usually considered to be at fault. Baldwin has stated that if the left half of the ventral bud persists and develops towards the left to join the main mass of the pancreas then an annular condition will result. Lecco suggests that the tip of the ventral anlage becomes adherent to the duodenal wall and in the normal migration becomes stretched to form a ring. If it is assumed that the ventral anlage is at fault, the annulus might be caused by an unusual development of the right bud or by the persistence of the left. In either case the duct should run antero-posteriorly from left to right; should the left bud be the cause of the condition the opening of the annular duct should lie posterior to the main duct. Both these facts were observed in the stereoscopic radiographs. Nearly all the cases so far recorded have an annular duct which empties into the main duct before the latter enters the duodenum. The present case appears to be unique in that it has an annular duct system which is separate and opens into the common bile-duct independently. The difference might be explained by the date at which the ventral bud becomes bifid, annular pancreas resulting if the bud persisted. In this present case it must either have existed as a separate bud or have become divided at a very early date. It is of interest to note the difficulty experienced in the injection technique, as the method previously recommended was bound to fail in a specimen which had a separate annular duct system.

CLINICAL FEATURES

McNaught gives an excellent review of 44 cases and therefore only a few points will be mentioned. The first case was described by Tiedemann in 1818. The majority of cases occurred in males, as did the present one. The age incidence shows great variety, as the youngest case was 3 days old (if Weissberg's embryo be excepted) and the oldest 74 years. Although the above case did not present any clinical symptoms, this is by no means always the case. The youngest case recorded died from intestinal obstruction caused by the annulus. In treating this complication gastro-enterostomy is the most frequent procedure. Howard reported a case in which division of the ring was followed by pseudocyst formation and drainage of the cyst resulted in a pancreatic fistula which healed spontaneously. However, the convalescence in this case was so stormy and prolonged that he came to the conclusion that a duodenojejunostomy would have been preferable. Many of the cases in the literature showed some other abnormality, but none were present in the above case. These abnormalities were of considerable variety and included abnormalities of the heart, aplasia of the kidney, club-foot, cleft palate, and accessory pancreas.

1. A case of annular pancreas unassociated with symptoms in a man aged 65 is described.
2. The origin of the abnormality is from the ventral bud, thus confirming the usual view.
3. Difficulties in injecting the duct system are described, these being due to an unusual condition of the annular duct.

I should like to thank Mr. H. N. Fletcher, senior surgeon to the hospital, for permission to publish this case; to Dr. L. R. Janes, the pathologist, for his help; and to both for their encouragement. I am also indebted to Mr. Stockham, radiographer, for several X-ray pictures taken at various stages of the investigation.

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OSTEO-ARTHRITIS OF THE HIP

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ALTHOUGH a considerable volume of literature has accumulated on the subject of osteo-arthritis of the hip the aetiology and treatment of this condition are still fruitful subjects of discussion. Better understood is its pathology, so ably studied by Nicholls and Richardson,[1] Lawford Knaggs,[2, 3] Timbrell Fisher,[4] and others. Further observations on the aetiology and treatment should, therefore, be of value not only with a view to ascertaining the best methods of treatment, but also of preventing this crippling condition.

In this paper is considered a consecutive series of cases of osteo-arthritis of the hip seen at the Wingfield-Morris Orthopædic Hospital and associated clinics since 1921. The number of cases studied was 242; in addition, 100 cases, picked at random and with no complaint of pain in the hip, were used as controls.

Incidence.—The average age at the onset of the symptoms was found to be 48·5 years for the whole series. The incidence of the age of onset in each decade in this series corresponds to most published figures.

The proportion of males to females was 6 to 5.

In 53 per cent of cases one hip was affected and in 47 per cent both hips.

The average age at the onset of unilateral disease was five years earlier than the age at the onset of bilateral disease (45 and 50 years respectively).

Questions concerning familial incidence were answered in 143 cases. In 20 per cent of these there was a suggestive family history of similar hip trouble.

Occupation.—Osteo-arthritis of the hip occurred in all types of occupations; 28 per cent of cases had spent most of their time at heavy work, 56 per cent at moderate work, and 16 per cent at light work.

Aetiology.—In the accompanying table (*Table I*) is given a list of the aetiological factors which appeared to lead to the production of osteo-arthritis, and the incidence of each.

Table I.—AETIOLOGICAL FACTORS IN OSTEO-ARTHRITIS

	Cases
Injury	66
Adolescent coxa vara	15
Protrusio acetabuli	12
Coxa plana	9
Septic arthritis	7
Dislocations of the hip	5
Rheumatoid arthritis	3
Childhood arthritis	3
Coxa vara (other than adolescent)	2
Acetabular deformity	2
Paget's disease of pelvis	2
Unknown—bilateral	97
Unknown—unilateral	19
TOTAL	242

A short discussion of each of these will, it is thought, be found helpful toward forming an opinion of their importance as well as their frequency.

Injury.—Injury may be an aetiological factor in the production of osteo-arthritis of the hip in several ways.

In the first group are those in which the injury was severe enough to result in a fracture of the acetabulum (with or without dislocation) or crush fracture of the head. An adult receiving such an injury will, as a rule, say that he has had pain ever since the accident and that this pain has been diminished by relief from weight-bearing. But once the hip is given normal use for some years, radiographs will reveal characteristic changes of osteo-arthritis. In this series there were 8 such cases. The majority had some form of reduction and extension treatment, usually followed by the use of a weight-bearing calliper.

In the second group are those in which the injury was not severe enough to result in fracture but osteo-arthritis resulted. There were 43 such cases. As in the first group, pain was continuous from the date of the accident. Of these, 50 per cent were free from arthritic signs in the opposite hip and 50 per cent had evidence of such disease. A proportion of the former probably come into the same category as the group with fractures and presumably had actual damage to the articular cartilage. The latter half requires further consideration. The other hip—although in most cases symptomless—demonstrated the three cardinal signs (loss of joint space, subchondral bone sclerosis, and new-bone formation) of osteo-arthritis on radiographic examination. Thus one may presume that the effect of the wear and tear of daily life had produced this symptomless background in both hips, and on the injured side the trauma had accelerated the process to the point of pain.

The third group consisted of those in which there was a time interval between the date of injury and the onset of arthritic pain. This group has a much more speculative aetiology. There were 15 such cases in the series under review, and one cannot be certain of the relation between trauma and arthritis.

Adolescent Coxa Vara (Slipped Epiphysis).—Slipping of the upper femoral epiphysis is frequently followed by osteo-arthritis of the hip. In this series there are 12 undoubted cases with typical histories and radiographic findings. In a further 3 cases the radiographic findings were characteristic, but a history was unobtainable.

In the accompanying table (*Table II*) it may be seen that the earlier the age of the slipping of the epiphysis the later the onset of osteo-arthritis. Thus the

Table II.—Slipped Epiphysis in Aetiology of Osteo-arthritis

Name	*Age of Slipping of Epiphysis*	*Age of Onset of Osteo-arthritis*
H. S.	12½	42
C. Haw.	14	30
C. Hus.	14	30
A. P.	14½	50
F. C.	15	36
C. P.	15	34
B. T.	16	28
T. W.	16	24
A. O.	16	24
A. B.	16½	25
M. Si.	16½	20
P. F.	17	19

youngest case (epiphysis slipped at the age of 12½ years) developed symptoms of osteo-arthritis at the age of 42, the oldest case (epiphysis slipped at the age of 17 years) developed symptoms at the early age of 19. The average age at the onset of osteo-arthritis was 32 years.

The slipped epiphyses in this series were treated by reduction when possible, followed by a period of fixation in either a plaster spica or Thomas splint with traction. Whether the modern method of reduction followed by the insertion of a Smith-Petersen pin[6] will delay the onset of osteo-arthritis, or even eliminate any predisposition to it, remains to be seen.

In all of the above cases the radiographic appearance of osteo-arthritis was so characteristic that one is justified in assuming such an aetiology when this appearance is seen (*see Fig.* 493).

FIG. 493.—Radiograph showing characteristic picture of osteo-arthritis superimposed on a previous slipped epiphysis.

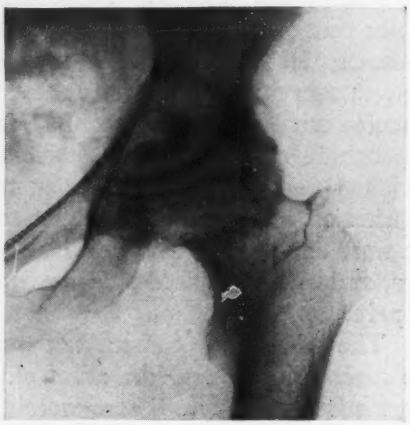

FIG. 494.—History of childhood limp, possibly rachitic type of coxa vara. Radiograph shows extreme loss of joint space, subchondral sclerosis, some osteophytic outgrowths, and cyst formation in the head (age 47).

Other Types of Coxa Vara.—Other less common types of coxa vara were encountered. One case of bilateral coxa vara had a history of a limp in childhood and osteo-arthritis beginning at 47 (*Fig.* 494). Another case suggestive of infantile coxa vara, with a history dating back to the age of 3, developed osteo-arthritis at the age of 30.

Coxa Plana.—There were 7 undoubted cases arising from this cause, with a typical history and radiographic picture. In 2 further cases the radiograph was typical, but the history of disability in childhood was not obtainable.

As in the cases of adolescent coxa vara, the same relation of the age of the disability in childhood to the age of onset of osteo-arthritis was apparent. Owing to the difficulty of obtaining accurate information about the age during which the coxa plana was active, these figures are not so striking as the previous series (*see Table III*).

The average age of the onset of the osteo-arthritic signs and symptoms in this group was 34 years.

Table V.—PERTHES' DISEASE IN AETIOLOGY OF OSTEO-ARTHRITIS

Name	*Age of Childhood Symptoms*	*Age of Onset of Osteo-arthritis*
F. B.	Unknown	46
Mrs. B.	5	47
H. W.	6	38
W. C.	Childhood	31
J. B.	Childhood	30
Mrs. D.	Unknown	30
Mrs. S.	7	26
F. W.	9	23
J. B.	9	21

Here again the radiographic appearance is so characteristic as to justify the diagnosis of the primary condition even when a history cannot be obtained (*Fig.* 495).

Protrusio Acetabuli (*Arthrokatadysis*).—This condition was clearly seen in 12 cases. In 3 the condition was bilateral and in 9 unilateral. Females were affected three times as frequently as males. Two unilateral cases had previously fractured the acetabulum, two unilateral cases gave histories of hip disease in childhood, and one bilateral case suffered from osteogenesis imperfecta.

FIG. 495.—History of Perthes' disease in childhood. Notice the characteristic long oblique upper margin of the shallow acetabulum fitting the very flattened head.

FIG. 496.—Arthrokatadysis, bilateral, of unknown cause. In addition the radiograph shows loss of joint space, subchondral bone sclerosis, and an osteophytic ring around the head. The medial wall of the acetabulum is thinner than usual.

When osteo-arthritis is associated with arthrokatadysis the medial wall to the acetabulum is thin, whereas in all other cases of osteo-arthritis it is thickened (*Fig.* 496).

Septic Arthritis.—In this group there were 7 cases, 2 of pneumococcal, 1 of streptococcal, and 1 of staphylococcal, origin. In the remaining 3 there was no mention of the organism concerned, but in one the history suggested a staphylococcal infection.

Congenital and Pathological Dislocation of the Hip.—There were 3 cases of congenital dislocation of the hip in this series. In 2 of these reduction had never been attempted and the age of onset of osteo-arthritis was 29 and 38 years. In these false joints subchondral bone sclerosis in the head of the femur is the predominating feature, but there is also loss of articular space in the false joints and

A

B

C

FIG. 497.—A, Radiograph taken 9 months after Lorenz reduction of congenital dislocation of the hip in a child (aged 7). B, Radiograph 14½ years after reduction (aged 21½). C, Radiograph 31 years after reduction (aged 38), showing slight loss of joint space, a small area of sclerosis, and osteophyte formation. (*Figs.* A *and* B *by courtesy of Mr. H. A. T. Fairbank.*)

there may be marginal lipping. The third case was a congenital dislocation reduced at the age of 7 (by Mr. Fairbank in 1907) by the Lorenz method, the age of onset of osteo-arthritis in this case being 34 (*Figs.* 497, A, B, C).

In one case of congenital subluxation of the hip the age of the onset of osteo-arthritis was 44. In one case of infantile paralysis a pathological subluxation of the hip at the age of 26 was followed by osteo-arthritis at the age of 28.

It would appear from the above that congenital dislocation of the hip may be followed by osteo-arthritis in the early thirties whether reduced or not.

Rheumatoid Arthritis.—A background of rheumatoid arthritis was evident in 3 cases, with many small joints affected during adolescence and early adult life.

Childhood Arthritis.—There were 3 cases of disease of the hip-joint in childhood the nature of which could not be ascertained. Two of the cases in this group showed marked distortion of the hip-joint.

Deformity of the Acetabulum.—In 2 cases a deformed acetabulum appeared to be the significant factor. In one case a developmental disturbance of the ischium had produced an alteration of the hip-joint mechanics; in the other, a cystic change in the pelvic bones associated with new-bone formation had apparently followed an injury.

Paget's Disease.—There were 2 examples of Paget's disease affecting one side of the pelvis with an associated osteo-arthritis of the hip on that side. In both of these cases the subchondral bone sclerosis was more marked than usual on the acetabular side, and in one case the rim of the acetabulum was so dense as to cut a groove in the head of the femur (*Fig.* 498).

FIG. 498.—Paget's disease affecting one side of the pelvis, with osteo-arthritis of the hip on that side.

Unknown Aetiology.—The number of cases in which a definite cause could be ascertained was 126—slightly more than half the total number investigated. Of the remaining 116 cases 19 were unilateral and 97 bilateral. One significant finding in the cases of unknown aetiology is that the age of onset of osteo-arthritis was a decade later than in those with an obvious cause. Generally speaking, then, if an obvious disorder of the joint has pre-existed the lesion is usually unilateral and of early onset; where there is no obvious cause the osteo-arthritis is usually bilateral and of late onset.

Shallow Acetabulum.—This factor has been given some importance, especially by continental writers.[8] It is true that in most osteo-arthritic hips the acetabulum is abnormally shallow. Measurements of the depth of the acetabulum were made by measuring the distance from a line joining the lateral margins of the superior and inferior rim of the acetabulum to the central portion of the acetabulum. The average depth of the acetabulum in 100 normal cases was 3·3 cm. and in 100 affected hips was 3·0 cm. In only 5 cases was the degree of shallowness such as is seen, for example, in coxa plana. Furthermore, if one analyses the measurements

of 100 normal and 100 affected hips it is found that the depth of the acetabulum plus the thickness of the acetabular floor is the same in osteo-arthritic cases as in the normal. Since these two totals are equal (*see Table IV*) one must assume that the increase in thickness of the floor of the acetabulum is equalled by the decrease in depth and is caused by deposition of new bone on the medial wall of the acetabulum.

Table IV.—CHANGES IN ACETABULUM IN OSTEO-ARTHRITIS

	Normal Hip	*Osteo-arthritic Hip*
Depth of acetabulum	3·3 cm.	3·0 cm.
Thickness of acetabular floor	1·1 cm.	1·4 cm.
TOTAL	4·4 cm.	4·4 cm.

Painless Osteo-arthritis.—In the course of this investigation 20 cases of osteo-arthritis were found in which the patient made no complaint either in the past or at the time of the examination; in every case the complaint was one of pain in the other hip.

Radiographic Features.—Three cardinal signs of osteo-arthritis were considered necessary before the diagnosis was established, i.e., loss of joint space, subchondral bone sclerosis, and the presence of osteophytes.

An attempt was made to correlate radiographic findings with the patient's symptoms. Pain cannot be measured and such evaluation could not be very accurate, but one was impressed by the fact that cases characterized by much new-bone formation were not as painful as those in which new-bone formation was minimal.

A B

FIG. 499.—A, In this radiograph the acetabular floor is 1·2 cm. in thickness. B, The same case 5½ years later; the acetabular floor is now 1·6 cm. This case also illustrates a layer of new-bone formation on the pelvic side of the acetabular floor.

New-bone formation on the lateral aspect of the acetabular floor was found in the majority of cases, a process that can be demonstrated in serial radiographs (*Fig.* 499). In *Fig.* 499, B the thickness of the acetabular floor has increased from 1·2 cm. (*Fig.* 499, A) to 1·6 cm. in $5\frac{1}{2}$ years. Most commonly occurring on the hip-joint side (*Fig.* 500), one occasionally finds new-bone formation on the pelvic side (*Fig.* 499, B). This new acetabular bone is always of a dense character.

FIG. 500.—In this case the acetabular new bone has been laid down on the lateral side, which is the usual finding. In this way the lower lip of the acetabulum is made more prominent.

FIG. 501.—Radiograph demonstrating the 'kissing' cysts. Subluxation and acetabular new bone may also be seen.

A B

FIG. 502.—A, Slight cyst formation. B, Same case 4 years later, showing marked cyst formation.

The thickening of the floor of the acetabulum occurring on the articular side may be partly responsible for subluxation of the head of the femur (*Fig.* 501).

Subluxation was observed in 92 cases. It is difficult to assess whether the subluxation was due to new-bone formation on the acetabular floor or whether the gradually increasing adduction produced the subluxation, which was then followed by deposition of new bone.

An inconstant feature which has roused much operative enthusiasm recently is the 'cyst'. They were found in 32 cases. The majority are found opposite the area of maximal loss of joint space and in the area of maximal bone sclerosis and here they are frequently of the 'kissing' type (*see Fig.* 501), but they may occur anywhere in the head or the acetabulum (*Fig.* 502, A, B), and may be either small or large. On further analysis of these 32 cases it was found that 25 complained of pain and 7 were painless.

TREATMENT

1. CONSERVATIVE TREATMENT

a. **Rest.**—This will nearly always relieve to some extent the pain of an osteo-arthritic hip. Patients commonly believe that if the joints are not kept on the move they will become completely stiff and are, therefore, afraid to rest as much as nature demands. To dispel such beliefs is a measure which may afford considerable comfort, and complete rest in a plaster spica is frequently followed by increased mobility. Rest may be afforded by a weight-bearing calliper. A weight-bearing calliper is useful when movements are relatively painless compared to the pain of weight-bearing itself, whereas a hip spica is useful when the pain is aggravated by movements. Some patients can be made comfortable by the alteration of their daily routine, while others may require complete rest in bed. Of great value in an adducted and flexed hip is the correction of the 'apparent' shortening by raising the shoe.

b. **Physiotherapy.**—This appears to benefit a minority, but is rarely of more than temporary benefit. Such benefit is lost if the patient has some distance to go for his treatment.

Deep X-ray therapy has been used for osteo-arthritis of the hip in this clinic for less than one year. The immediate results are promising in 12 out of a total of 15 patients receiving this therapy. Whether this effect is lasting remains to be seen. Dausset and Lucy[9] reported definite improvement in half of their cases for periods up to two years (total of 50 cases). Watt[10] claimed a higher percentage of satisfactory results, especially in early cases.

2. OPERATIVE MEASURES

When conservative measures fail and an otherwise fit patient is prevented from carrying on his work or daily routine, operative treatment must be considered. Such procedures include manipulation, cheilotomy and synovectomy, reconstruction operations, formal pseudarthrosis, excision of the head and neck of the femur, subtrochanteric osteotomy, arthrodesis, and drilling of the femoral neck. Descriptions of each of these procedures will not be attempted in this paper and the following remarks will be confined to the estimation of results obtained.

a. **Cheilotomy and Synovectomy.**—These procedures together were employed in the treatment of 2 cases, both of which were improved—one sufficiently to allow the patient to return to a heavy occupation.

Cheilotomy alone was performed in 5 cases. Of these, 2 were failures—although one was temporarily improved, other operative measures were required later; 2 were moderately improved; and 1 was greatly benefited and was able to return to heavy work.

b. **Reconstruction Operations.**—Following the technique of Whitman,[11, 12] the operation of reconstruction was performed on 9 hips. In a further 4 cases the operation was done without transplanting the greater trochanter. In 3 cases there was an increase in the range of movement of 5–20 per cent. In the remaining 10 cases the movements were the same or diminished as a result of the operation. In 6 of the 13 cases the pain was abolished and in 2 cases the pain was lessened. In the remaining 5 cases the pain was not altered by the operation, and in 2 of this latter group an arthrodesis was performed later.

Thus satisfactory results as far as pain was concerned were obtained in less than half the cases, whereas a good range of movement was not obtained in any one case.

c. **Formal Pseudarthrosis.**—There were three types of operation used which may conveniently be described under this heading.

In the first the neck is excised, the head being left in the acetabulum, the greater trochanter then being transplanted to the medial side of the upper end of the femur so that its raw surface comes against the raw medial side of the trochanteric region. There were 2 such operations. One was very successful in that movements were increased from nil to 70 per cent and the patient was free from pain. The other case was followed by pain and decrease in movements and required further operative treatment. In both there was considerable resultant shortening (3 in. and 3½ in.).

In the second procedure the head and neck of the femur were excised and the greater trochanter again was transposed to the medial side. There were 5 cases dealt with in this manner. In 3 of these the result was a marked increase in movement and freedom from pain. In 1 case the movements were not improved but the pain was lessened, and 1 case was a failure.

The third procedure consisted of excision of the head of the femur, with or without remodelling of the neck and moving of the greater trochanter farther down the shaft. Two patients had this type of operation. In one the movements were lessened, but there was freedom from pain, and the other was a failure which required further operative measures.

d. **Excision of the Head and Neck of the Femur.**—This operation, first done by Fock[13] in 1857, consists of excision of the head and neck of the femur and, when necessary, removal of the projecting rim of the upper edge of the acetabulum. It was employed in the treatment of 23 cases. In every case there was an increase in the range of movement, frequently as much as 60 per cent. Three patients complained of occasional pain in the hip, but volunteered that they were much better than before operation, and all the rest of the patients were entirely free from pain.

These patients, after a post-operative traction of six weeks in a Thomas splint, were allowed to walk in a weight-bearing calliper. The calliper was usually employed for from one to two years and then gradually left off, and some of the patients were able to carry on with their previous occupation (light work) without wearing an instrument, but with a lift on the shoe. The average pre-operative

shortening was ½ in. and post-operative 1⅝ in., so that a lift was usually advised. The average time off work for this whole group was six months.

There were no post-operative deaths.

e. **Subtrochanteric Osteotomy.**—This procedure was used by itself in 4 cases and resulted in only temporary or partial relief in each case.

f. **Arthrodesis.**—Arthrodesis was performed on 23 patients. The combined intra- and extra-articular method was used in 16 cases. Of these 9 were freed from pain, had a clinically sound ankylosis, and in all but 1 case there was sound bony ankylosis by radiographic examination. Of the remaining cases, 5 were definitely improved but still had slight pain, and in these there was no ankylosis demonstrated either clinically or radiographically. In 1 patient the operation failed to relieve pain or to procure ankylosis, and was therefore repeated; it is still too early to estimate the result in this case. There was 1 death (post-operative collapse, death after 5 days). The extra-articular graft alone was used in 7 cases, and all but 1 of these patients were completely freed from pain, although only 3 had X-ray evidence of bony ankylosis.

Twelve patients went back to work and the average time off work following arthrodesis was 19 months.

Only 1 case in this series developed low back pain following successful arthrodesis.

In 48 per cent of all cases of arthrodesis there was radiographic evidence of bony ankylosis.

g. **Drilling of the Femoral Neck.**—In only 1 case in this series was this procedure adopted; there was only temporary benefit.

DISCUSSION ON AETIOLOGY

From the study of this series of cases, the theory that osteo-arthritis is an end-result of wear and tear appeared most plausible. It was Pommer[14] who enunciated this 'functional' theory of causation of osteo-arthritis. Normal use of a joint through a life of average length, and with little damage by toxins, bacterial infection, or trauma, would result in a joint which indeed would show some evidence of wear and tear, i.e., fibrillation of cartilage, but still with an intact covering of cartilage, a state of affairs which would rarely cause the patient pain.

A condition which alters the anatomy of any part of the hip-joint will accelerate the effects of wear and tear of use, so that osteo-arthritis results. We have already seen that injury is the most common cause of osteo-arthritis; an alteration of the normal congruity of the head and the acetabulum due to slipped epiphysis, arthrokatadysis, or coxa plana comes next in order of frequency.

As demonstrated in the series of slipped epiphysis and to a less degree the series of coxa plana, osteo-arthritis was of relatively early onset when the primary condition was of late onset. In other words, adaptation of the shape of the acetabulum to the new form of the head takes place more effectively in the more tender age periods. This is further illustrated by comparing the radiographs of these cases, e.g., in an early-age coxa plana with a late onset of osteo-arthritis the upper margin of the head and neck is long and flat and is fitted exactly by a long flat roof to the acetabulum. It is reasonable to suppose that the closer the conformity of the acetabular shape to the shape of the femoral head the more delayed

is the onset of osteo-arthritis. The degree of conformity of the acetabulum to the deformed head will be directly proportional to the age of onset of the primary condition.

Generally speaking, the higher the age group, the greater the percentage of cases with osteo-arthritis. The findings of Heine[15] and Beitzke[16] suggest that some degree of osteo-arthritis is universal after the age of 50. But such anatomical findings include many cases that demonstrate neither clinical nor radiological evidence of this condition, and the effects of wear and tear are only apparent in the anatomical specimen. Unilateral cases occur at an earlier age than bilateral cases[17] and in most a cause can be found. Bilateral osteo-arthritis occurs at a later age and the majority are due to the effects of wear and tear.

FIG. 503.—Case of Charcot hip. Osteo-arthritis with marked overgrowth and distortion, but painless. This patient was a syphilitic. (*By courtesy of Mr. Nicholson.*)

The question of painless osteo-arthritis is an interesting one. The explanation may be as follows: Newman and Corbin,[18] Corbin and Gardner,[19] Inukai,[20] Pearson,[21] and others, have demonstrated that degeneration in the central nervous system increases with advancing years. If painful stimuli are diminished or abolished by such a process, a condition exists which is to some extent comparable to a neuropathic joint. In such a joint the increasing insensitivity permits of more freedom of use and therefore the joint is subjected to further traumata. It was pointed out earlier in this paper that the cases with much new-bone formation were often relatively painless. The analogy is complete when it is found that a true neuropathic joint may be radiographically identical with many of these painless joints (*Fig.* 503).

DISCUSSION OF OPERATIVE TREATMENT

Unilateral osteo-arthritis of the hip in the young patient calls for treatment which will give freedom from pain and a stable joint. Such a result should be obtained from arthrodesis.[5, 22, 23] Stability was maintained in all our cases; pain was completely relieved in 65 per cent, and partially relieved in a further 22 per cent. Sound ankylosis could perhaps be more regularly obtained by the method described by Watson Jones,[24] i.e., intra-articular arthrodesis, supplemented by a pin transfixing the joint. The time off work could almost certainly be lessened by this means.

Although patients can get about with two stiff hips, good movement in one is most desirable. If pain is present, a procedure which relieves pain and allows mobility is particularly indicated. Reconstructive operations aim to achieve this, but the results in this series are disappointing. Arthroplasty has failed in the hands of most surgeons. The results of excision of the femoral head and neck, however, are uniformly good. It is true that some stability has been lost and that a shortening averaging 1½ in. is produced. But almost all the patients subjected to this operation were completely relieved of pain and developed a good range of movements. The average time off work was six months, and though all of these patients went back to light work only, their period of incapacity was considerably shorter than that for arthrodesis. The amount of stability that may be achieved may be judged from the fact that the majority of these patients began to do without their callipers after about eighteen months and were walking well and with only a slight limp. This operation is also of value to patients with one hip affected and whose occupation entails light work.

Subtrochanteric osteotomy is a relatively safe procedure which gives stability and is said to relieve pain. McMurray[17] reports good results, and although the small experience of the operation at Oxford has not been encouraging, the writer feels that it should be given a more extensive trial.

Drilling the femoral neck for the 'relief of tension' in the cyst-like cavities has had variable success; MacKenzie[25] reported good results in 80 per cent of cases, Boppe[26] 50 per cent, Simpson and Henderson[27] 50 per cent, and Ducker[28] 10 per cent. It has already been mentioned that cysts were seen in 32 cases and in 7 of these the hip was painless. Thus it can be said that pain is not always coincident with the presence of cysts, and further investigations are necessary in order to determine which cases will benefit from this operation.

CONCLUSIONS

1. In slightly more than half the 242 cases a definite cause was found, and the majority of theses cases were unilateral.

2. Aetiological factors, in order of frequency were: injury, slipped epiphysis, arthrokatadysis, coxa plana, septic arthritis, dislocations, various forms of arthritis, coxa vara (other than slipped epiphysis), acetabular deformity, and Paget's disease of the pelvis.

3. The earlier the onset of slipped epiphysis and coxa plana, the later the onset of osteo-arthritis.

4. Bilateral cases occur at a later age period than unilateral, and the cause may be ascribed to the wear and tear of use.

5. Conservative treatment with the aim of resting the joint frequently results in considerable, though perhaps temporary, improvement. Physiotherapy afforded only temporary benefit, whereas the results of deep X-ray therapy are encouraging.

6. Operative measures of value include arthrodesis and excision of the femoral head and neck.

The author wishes to express his gratitude to Lord Nuffield, through whose generosity this clinical study was possible, and to Professor G. R. Girdlestone, whose valuable criticisms and suggestions have been of inestimable help.

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THE SURGICAL TREATMENT OF MID-ŒSOPHAGEAL CARCINOMA

By HAROLD WOOKEY, Toronto

The results of the radiological treatment of œsophageal carcinoma have proved disappointing and a new interest in the possibility of the surgical excision of such tumours has arisen.

The high operative mortality in the surgical removal of the œsophagus has led to the general adoption of palliative measures, but many of these unfortunate patients would readily agree to a radical operation if there were a fair chance of survival.

Carcinoma of the œsophagus is relatively slow in its growth and the spread to regional lymph-glands occurs late in the disease.

The present paper deals only with the mid-œsophageal growths. They are usually situated at about the level of the left bronchus and partly behind the arch of the aorta. The earliest symptoms of the condition are difficulty in swallowing and substernal discomfort or pain. Unfortunately, the diagnosis is often delayed until there is marked weakness and loss of weight.

The first step to be taken is to establish the diagnosis both by radiological means and by the use of the œsophagoscope with biopsy.

Some form of gastrostomy must then be done and the patient's condition improved by judicious feeding and blood transfusions.

The conventional method of approach for removal of the œsophagus has been through the left side of the chest, but I have found that the right-sided approach possesses many advantages and renders the operation much simpler.

This paper is based on 4 cases in which the thoracic œsophagus was removed.

In the first case the usual approach through the left side of the chest was employed, but in the last three the exposure was through the right side of the chest. It was found by operations on the cadaver that by the division of the vena azygos major close to its junction with the superior vena cava, the right lung, together with other mediastinal structures, could be readily displaced forwards and to the left.

The œsophagus could then be completely exposed and most of the technical difficulties eliminated.

OPERATIVE TECHNIQUE

The following description, illustrated by drawings made in the operating room, demonstrates the technique and steps in the operation.

The first step is some form of gastrostomy. The Janeway tube gastrostomy was used in three cases and a simple jejunostomy in the fourth. A tube of the entire thickness of the anterior wall of the stomach was prepared with its base along the lesser curve, the free end stitched to a suitable opening in the skin below the sternum. (*Fig.* 504.) The patient is allowed to recover from this operation

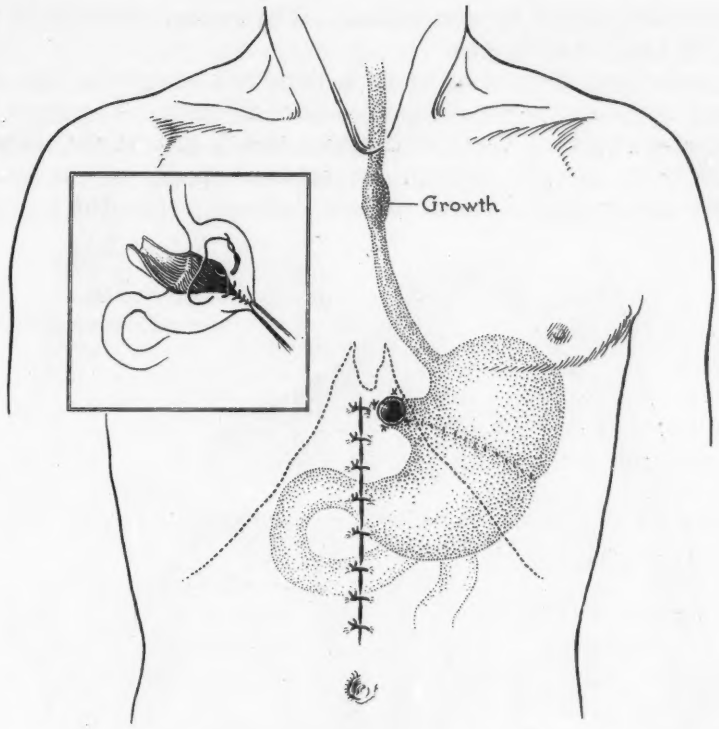

FIG. 504.—The Janeway tube gastrostomy completed. *Inset* shows the formation of the tube from the anterior portion of stomach wall.

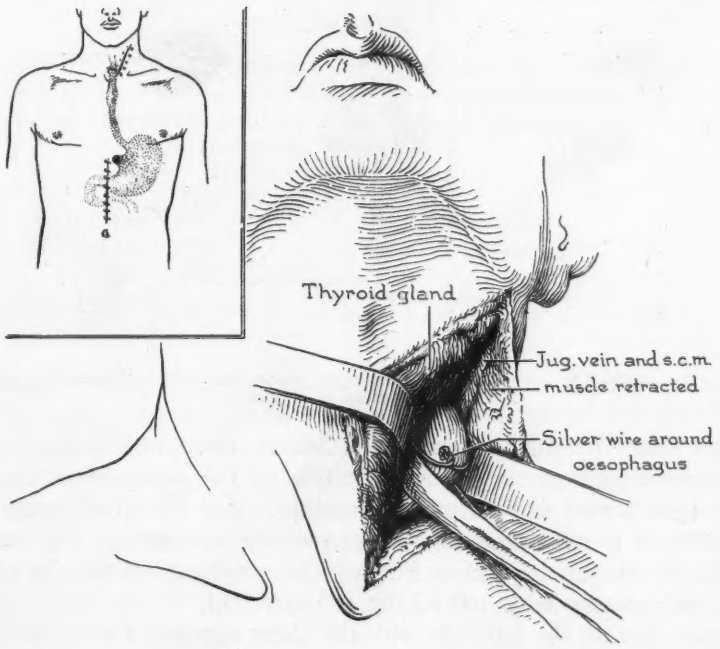

FIG. 505.—Exposure of upper two inches of œsophagus and application of silver wire *Inset* shows wire in place and neck incision closed.

and to become accustomed to tube feeding. The general condition of the patient is improved by blood transfusions.

Three weeks later the second stage is done and consists of the exposure of the upper end of the œsophagus in the left side of the neck. The upper two inches is mobilized and a loop of silver wire passed loosely around the œsophagus, the twisted ends being brought through the anterior margin of the sternomastoid muscle. The neck wound is closed without drainage. (*Fig.* 505.)

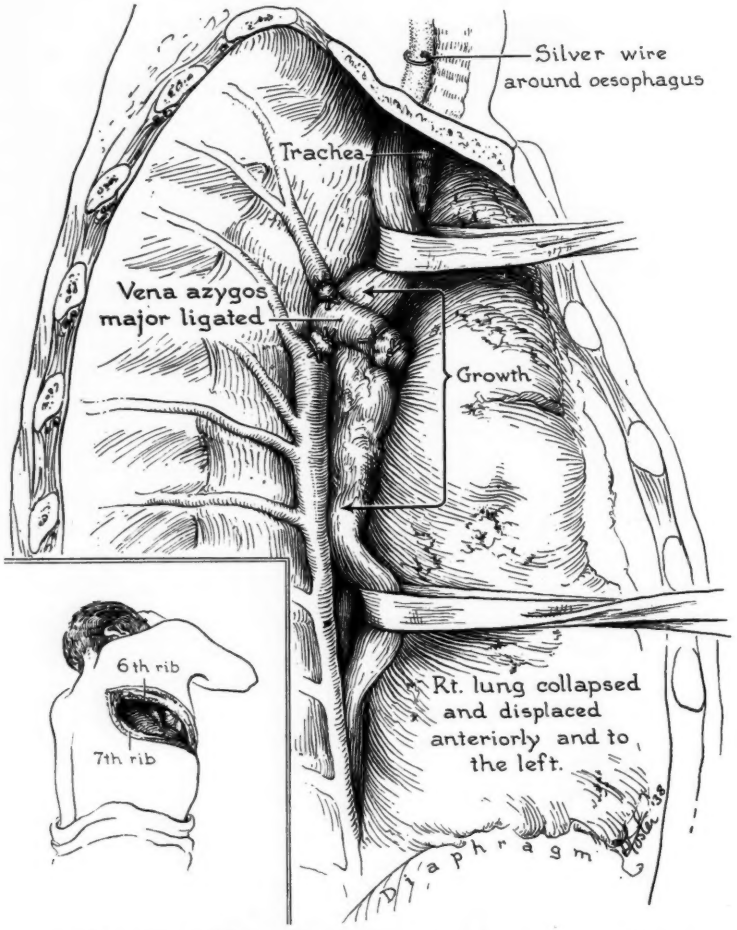

FIG. 506.—The isolation of the œsophagus above and below the growth. *Inset* shows method of approach.

Five days later the third operation is done. The previous exposure of the upper end of the œsophagus cuts down the time of the major procedure considerably and has been found to be a great advantage. For the third operation, intratracheal anæsthesia is used—gas and oxygen or cyclopropane. The intratracheal catheter is fitted with an expanding bag which completely closes the trachea and in this way the pressure within the lung is controlled.

The patient lies on the left side with the chest supported by a sandbag. The arm is drawn forwards and upwards and secured to an arm-rest. A continuous drip transfusion is given throughout the operation.

A long incision is made over the seventh interspace just below the angle of the scapula. The seventh and eighth ribs are divided as far back as possible, the pleura opened along the line of incision, and heavy self-retaining retractors spread the ribs, affording ample space for the further steps of the operation. Any pleuritic adhesions are divided, and with the collapse of the lung the vena azygos major is easily seen as it turns forward to enter the superior vena cava. The vena azygos major is divided between ligatures. The right lung, together with the mediastinal

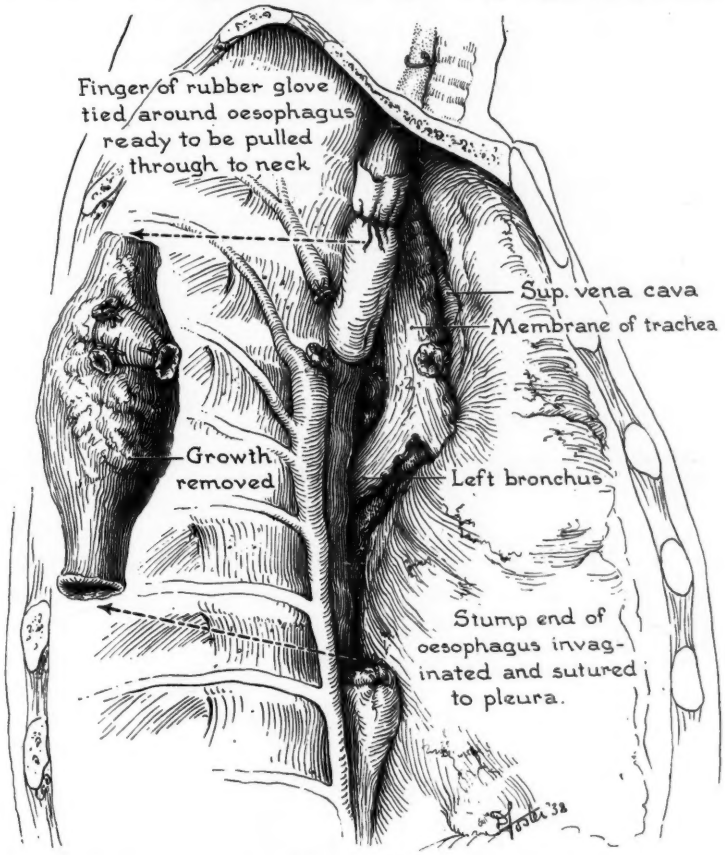
Finger of rubber glove tied around oesophagus ready to be pulled through to neck
Sup. vena cava
Membrane of trachea
Growth removed
Left bronchus
Stump end of oesophagus invaginated and sutured to pleura.

FIG. 507.—Showing the method of dealing with the upper and lower ends after the œsophagus has been divided. The middle tumour-bearing portion was in this case resected, as, owing to its bulk, contamination was feared.

contents, is now displaced forwards and to the left. A long incision is made through the mediastinal pleura along the right margin of the œsophagus. This incision is carried down to the diaphragm. The œsophagus is now freely exposed, together with the tumour, and one may quickly decide whether removal is possible. The œsophagus is freed above and below the tumour mass, tapes being passed around it, above and below. (*Fig.* 506.) By blunt dissection the tumour is completely freed from surrounding structures, great care being used to protect the left bronchus and pleura. Should the left pleura be opened, the opening is at once closed.

After completely separating the tumour from the surrounding structures, the œsophagus in the upper part of the thorax is easily freed by blunt dissection, and

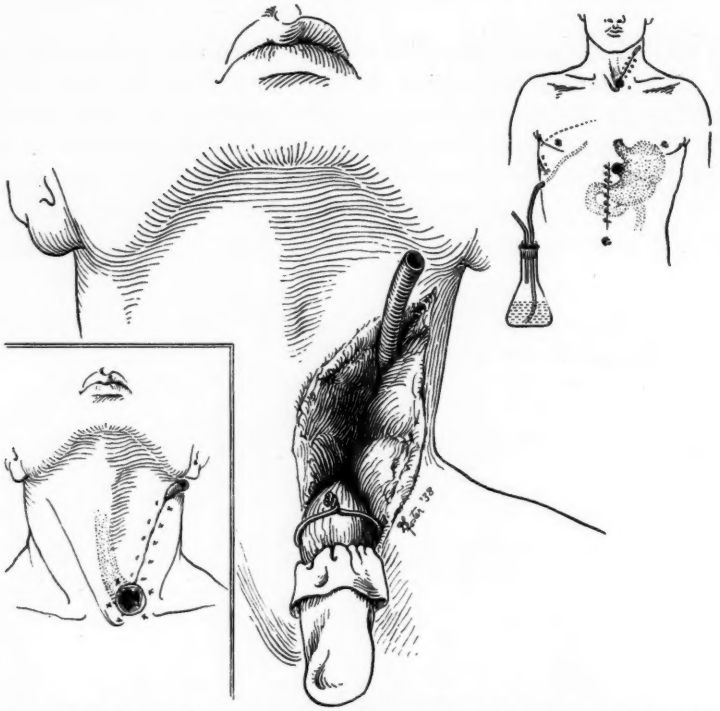

FIG. 508.—Showing the œsophagus pulled through the neck incision and sutured to the skin. *Insets*: *Top*, Closed drainage of chest, operation completed; *Bottom*, Circle of skin removed to receive end of œsophagus.

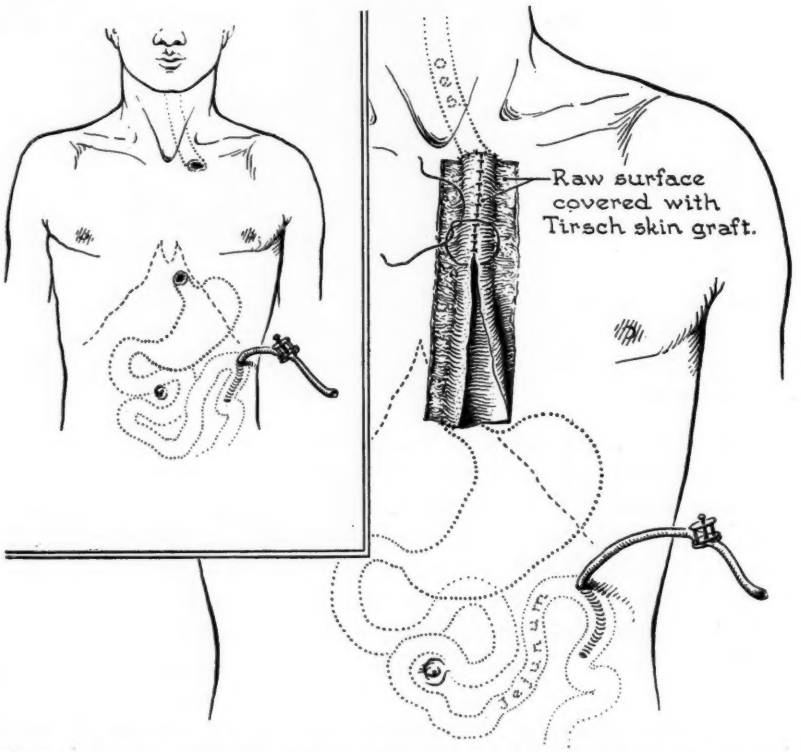

FIG. 509.—Diagram illustrating method of making new œsophagus from skin of chest wall. *Inset* shows end of œsophagus brought out and sutured to skin; jejunostomy tube retained.

the loop of silver wire felt. This may all be done without seeing the aorta or any of the great vessels and is very much simpler than the same procedure from the left side of the chest. The œsophagus is now divided as low down as possible by cautery, the lower end ligated and invaginated, and this is allowed to slip beneath the pleura. The upper cut end of the œsophagus is ligated and a finger stall drawn over it and secured by ligatures, to prevent contamination. (*Fig.* 507.) The opening in the mediastinal pleura is now closed and the chest wound closed by interrupted sutures passed around the ribs. The chest is drained by closed drainage introduced through a stab wound in the tenth interspace in the mid-axillary line. The patient is now turned on his back, the neck wound opened, the silver wire quickly identified, and the œsophagus brought out through the opening in the neck (*Fig.* 508).

In the case illustrated by the diagrams, the œsophagus was divided above the growth, which was rather bulky, and one was afraid of contamination, but this is not usually necessary. The œsophagus is divided at a favourable position and the cut end brought through the lower end of the incision. If long enough, it is brought out through a tunnel beneath the skin, and if possible below the clavicle. The cut end of the œsophagus is carefully sutured to the skin and the neck wound closed with a small drain.

RECONSTRUCTION OF THE OESOPHAGUS

Following the removal of the thoracic œsophagus a patient may live indefinitely with a gastrostomy. In some cases the upper end of the œsophagus may be connected to the lower opening by a rubber tube. This method was tried in *Case* 2, but it was found almost impossible to prevent a leak, particularly from the upper end. Consequently, an attempt was made to reconstruct the œsophagus by means of a tube of skin on the chest wall. This tube was successfully made, but healing at the lower end did not take place because of the erosive effect of gastric secretion.

To overcome this difficulty, in *Case* 4 a preliminary jejunostomy was done in the first stage so as to preserve the stomach intact for further use. The patient, having recovered from the removal of his œsophagus, was again operated upon. The abdomen was opened through a small oblique incision just below the left costal margin. The lower end of the œsophagus was exposed and freed as it passed through the diaphragm. It was found to be about three inches long and its invaginated end was easily brought to the surface and sutured to the skin just below the costal border. It was not necessary to divide the left gastric artery or to disturb the stomach in its normal relations. The wound healed readily and it was found that there was no escape of gastric contents from the œsophagus, probably because the cardiac sphincter still functioned.

A tube of skin was made as shown in the diagram (*Fig.* 509) and the upper and lower connexions with the œsophagus were made without difficulty. The patient was fed through the jejunostomy until healing had taken place, after which the jejunostomy tube was removed. At first fluids only were given by mouth, but it was soon found that the patient could take food of any description. He has returned to work and has gained nearly 30 lb. in weight. A radiograph taken after swallowing barium shows the position of the new œsophagus (*see Fig.* 514).

This method of reconstructing the œsophagus has proved effective and is not particularly difficult. It removes the great disability and the depressing effect of a permanent gastrostomy.

THE COLLATERAL CIRCULATION AFTER THE LIGATION OF THE VENA AZYGOS MAJOR

The ligation of the vena azygos major in the right-sided approach to the œsophagus has not produced any apparent ill effect in the cases here reported. Through the kindness of Professor J. C. B. Grant, of the Anatomy Department, University of Toronto, the path of the collateral circulation has been demonstrated and he has been good enough to make the following comments: "The question may be asked, 'By what paths does the blood find its way back to the heart after the arch of the azygos vein has been ligated?' It has been shown in the cadaver by means of retrograde injections, reversing the flow in the azygos vein, that ready exit is found by several of the following channels, the selected channels varying from specimen to specimen with the varying patterns of the veins: (1) Downwards via the azygos vein (which passes through the right crus of the diaphragm) to communicate with the inferior vena cava at the level of the right renal vein; (2) Across the front of the vertebral column via the (left) hemiazygos veins, of which there are one, two, three, or more, thence (*a*) downwards via the inferior hemiazygos vein (which passes through the left crus) to communicate with the left renal vein, and (*b*) upwards via the superior hemiazygos vein and onwards via the left superior intercostal vein to the left innominate vein and so to the superior vena cava; (3) Downwards via the right and left ascending lumbar veins (situated behind the psoas muscle) which connects the lower intercostal veins with the lumbar veins and with the common iliac veins; (4) Backwards via the intercostal veins, thence via their intervertebral branches (which pass through the intervertebral foramina) to communicate with the vertebral plexus of veins. This plexus lies within the vertebral canal and consists of several cavernous-like, longitudinally running channels which extend from the foramen magnum to the sacrum."

CASE REPORTS

Case 1.—B. B., male, aged 51. Admitted to hospital January 13, 1937.

HISTORY.—*Constant* discomfort beneath lower end of sternum—1 year. Weakness and loss of weight—3 months. Pain and difficulty in swallowing—6 weeks.

ON EXAMINATION.—

X rays.—A defect in the œsophagus 1½ in. long along the posterior wall of the œsophagus. Very little obstruction. Defect at level of left bronchus. (*Fig.* 510.)

Œsophagoscopy (Jan. 14).—A growth involving the anterior wall of the œsophagus, 30 cm. from the teeth. Biopsy: Epidermoid carcinoma.

1ST OPERATION (Jan. 14).—Janeway gastrostomy.

Jan. 31.—Several small hæmorrhages from the œsophagus.

2ND OPERATION (Feb. 16).—Left-sided approach. No pleuritic adhesions. Tumour adherent to arch of aorta in its upper third. Appeared to be about 2½ in. long. Tumour adherent to right pleura. Right pleura accidentally torn; opening closed. Œsophagus divided below and invaginated. Œsophagus exposed in left side of neck. Entire œsophagus and tumour drawn through neck wound. Divided and sutured to skin below clavicle. Chest wound closed. Closed drainage. Continuous blood transfusion. Patient died in 48 hours, apparently as the result of coronary thrombosis.

PATHOLOGIST'S REPORT.—Tumour almost completely encircling the œsophagus. Microscopically an epidermoid carcinoma, invading wall of œsophagus. Regional lymph-glands showed inflammation only.

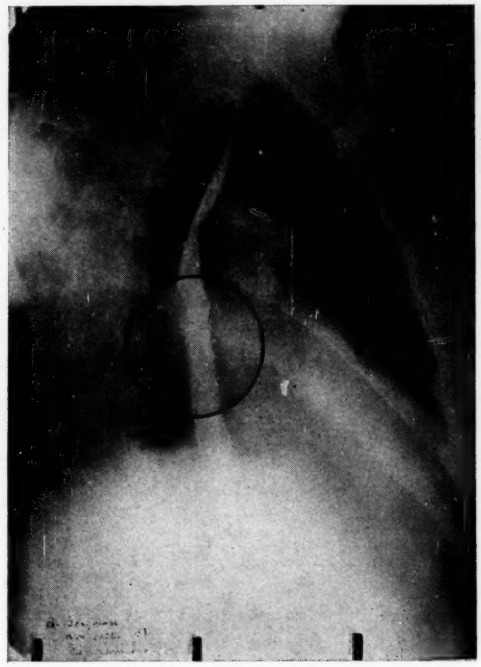

FIG. 510.—*Case* 1. Lateral radiograph showing defect along the posterior wall of œsophagus, at level of left bronchus, but very little obstruction.

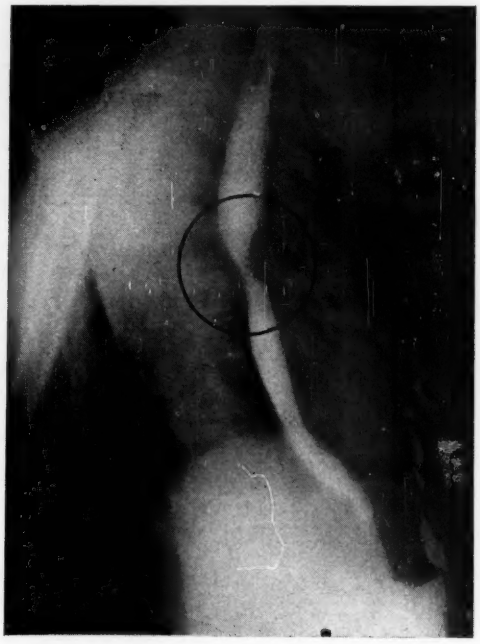

FIG. 511.—*Case* 2. Radiograph showing œsophageal obstruction at level of C. 7, 8.

Case 2—F. M., male, aged 49. Admitted to hospital April 11, 1937.

HISTORY.—Burning sensation behind middle of sternum on swallowing—3 months. Difficulty in swallowing solid food—3 months. Lost 20 lb. in weight in last 2 months.

ON EXAMINATION.—

X rays.—An obstructive lesion in œsophagus at level of 7th and 8th thoracic vertebra (*Fig*. 511).

Œsophagoscopy (April 17, 1937).—Biopsy: Epidermoid carcinoma.

1ST OPERATION.—Janeway operation.

2ND OPERATION (May 6, 1937).—Exposure of œsophagus, left side of neck, passing loop of silver wire.

3RD OPERATION (May 10, 1937).—Excision of œsophagus. Right-sided approach.

PROGRESS.—This patient made a good recovery and lived for two years. He apparently died of secondary carcinoma of the lung. No autopsy obtained.

PATHOLOGICAL REPORT.—Tumour mass almost completely surrounding œsophagus, with marked obstruction. Microscopically an epidermoid carcinoma invading wall of œsophagus. Regional lymph-glands show inflammation only.

Case 3.—H. R., male, aged 56. Admitted to hospital Nov. 14, 1937.

HISTORY.—Difficulty in swallowing solid food—5 weeks. Substernal discomfort—2 months. Loss of weight, 10 lb. in 2 months.

ON EXAMINATION.—

Œsophagoscopy (Oct. 27, 1937).—An annular tumour at level of 5th and 6th thoracic vertebra about 1 in. in length. Biopsy: Epidermoid carcinoma.

X rays.—An annular filling defect middle third of œsophagus. Practically no dilatation of œsophagus above lesion (*Fig*. 512).

1ST OPERATION (Nov. 9, 1937).—Janeway gastrostomy.

2ND OPERATION (Dec. 2, 1937).—Exposure of œsophagus left side of neck. Passing of loop of silver wire.

3RD OPERATION (Dec. 7, 1937).—Removal of œsophagus—right-sided approach. Growth found to be adherent to left bronchus, from which it was separated with some difficulty.

PROGRESS.—Died on Dec. 11, 1937. Partial autopsy showed perforation of posterior membranous wall of left bronchus, with infection of pleura and mediastinum.

PATHOLOGICAL REPORT.—Tumour almost completely encircles œsophagus. Microscopically an epidermoid carcinoma, infiltrating the submucosa and at one point the muscle coat. Lymph-nodes showed no evidence of carcinoma.

Remarks.—In this case, on closing the chest wound the collapsed lung was immediately expanded through the intratracheal tube, and it is felt that this was a mistake, and might have contributed to the perforation of the left bronchus which was found at autopsy.

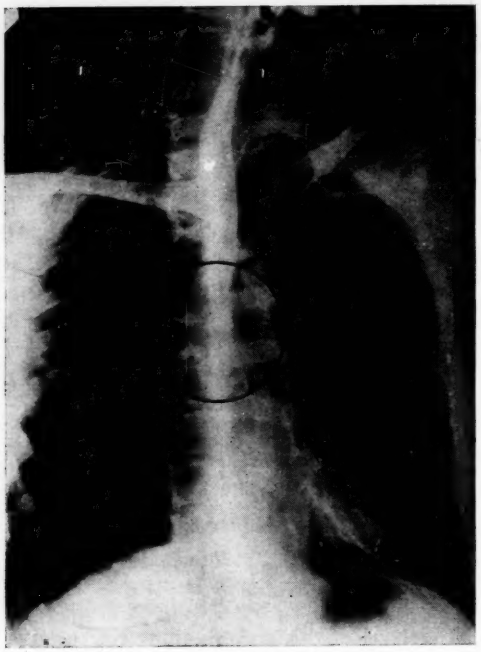

FIG. 512.—*Case* 3. Radiograph showing filling defect in middle third of œsophagus.

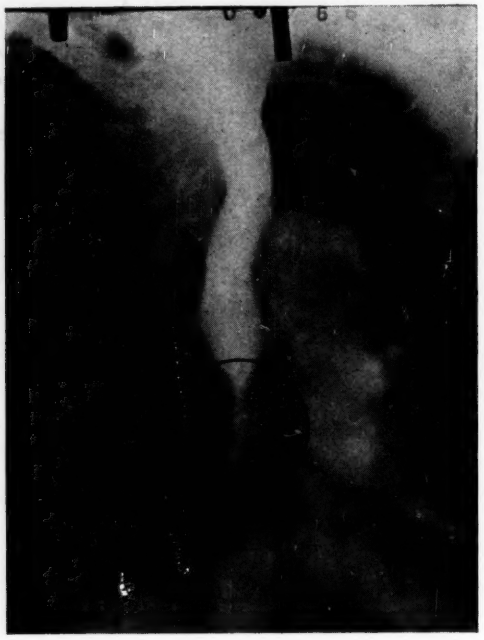

FIG. 513.—*Case* 4. Radiograph showing constriction of œsophagus in region of left bronchus and moderate dilatation above.

Case 4.—J. D., male, aged 62. Admitted to hospital April 27, 1938.

HISTORY.—Increasing difficulty in swallowing—6 months. Marked loss of weight.

ON EXAMINATION.—

X rays.—Constriction of œsophagus, region of left bronchus. Moderate dilatation of œsophagus above. (*Fig*. 513.)

Œsophagoscopy (May 3, 1938).—Constricting tumour, mid-thoracic region. Biopsy: Epidermoid carcinoma.

1ST OPERATION (May 3, 1938).—Jejunostomy done about 12 in. from duodeno-jejunal flexure.

2ND OPERATION (May 13, 1938).—Exposure side of œsophagus left of neck. Loop of silver wire introduced.

3RD OPERATION (May 25, 1938).—Excision of œsophagus through right side of chest.

4TH OPERATION (July 11, 1938).—Through an oblique left upper abdominal incision the lower cut end of the œsophagus was freed from the diaphragm and brought out through the abdominal wall and sutured to an opening in the skin just below the costal margin, as the permanent gastrostomy.

5TH OPERATION.—The œsophagus was reconstructed as described on page 701.

PROGRESS (August, 1939).—The patient has made a good recovery and is apparently quite well. Radiographs of the chest show no evidence of disease (*Fig.* 514). He has gained over 25 lb. in weight.

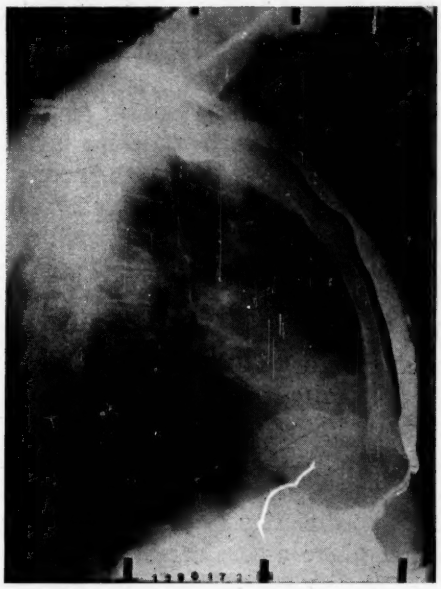

FIG. 514.—*Case* 4. Radiograph showing position of reconstructed œsophagus. There is no evidence of disease in the chest.

PATHOLOGICAL REPORT.—An ulcerating carcinoma almost entirely surrounding the œsophagus. There were several small lymphatic glands.

Microscopically, an epidermoid carcinoma invading the submucosa down to the muscle coat. The lymph-glands showed no evidence of carcinoma.

I am very much indebted to Dr. Keith Welsh for his valuable assistance, and to Professor Grant for his anatomical contribution.

'BIRTH FRACTURE' OF THE TIBIA

By BRYAN McFARLAND

I AM describing a method for obtaining union of 'birth fractures' of the tibia. I do not think it is original, but a search made for my source of inspiration has been unsuccessful. The method, however, is both successful and simple. I retain the name 'birth fracture', though I doubt its accuracy. For why should this fracture fail so persistently to unite when 'nothing will stop' other birth fractures from uniting? And how seldom at birth is the tibia seen fractured in this position, compared with the femur, the humerus, or the clavicle!

Any surgeon who has had to deal with the condition known as 'birth fracture' (or 'congenital pseudarthrosis') of the tibia will realize the difficulty of obtaining sound bony union. This difficulty is illustrated in at least two of the cases which I record below. As recently as May, 1939, the President of the British Orthopædic Association speaking on bone-grafts said that "no satisfactory way of grafting birth fractures of the tibia has been found". Why there should be this difficulty I do not know for there is no difficulty in promoting union by bone-graft in other forms of non-union. Even where a considerable gap exists it can be bridged and solid bony union results. As an example of the difficulty of obtaining union in birth fractures, I can remember many years ago seeing a surgeon perform what he told me was the fifteenth operation on a girl's leg to try to make the bones unite. I can also recall a young adult's leg being finally amputated because there was still non-union and the limb was so shortened and atrophic that, even if union had been eventually obtained, the surgeon thought the leg would be of very little use. There is no doubt that persistence of non-union does seriously impair the growth, and if by ten years of age there is still non-union, the tibia is inevitably much shortened.

From observations of my own cases one might almost think that something more than the mechanical factor is responsible for maintaining non-union. It is as if there were some process of bone absorption carried out by cells actively destroying any bone which may form or be placed in the area, and reducing it to fibrous tissue and cysts. But it will be seen that after internal fixation by a 'side' strut the fracture unites, so why it should not do so with efficient external fixation is incomprehensible.

I remember reading that if a child with tibial pseudarthrosis were encountered early and the limb immobilized continuously for a sufficiently long time, union would result. In my third case it will be seen that this policy was pursued. For a period of four years the limb was never out of plaster. On the lines advocated in the article I read, changes of plaster were made in half stages so that the limb was never for a moment unsupported. It will be seen that nevertheless union did not occur, although immobilization was augmented by an ordinary inlay bone-graft (*see Figs.* 526, 527).

Two methods of obtaining union are indicated, but I advocate the second because, as will be seen from the accompanying radiographs and accounts of the cases, it is practically certain and it is simple.

Of the three cases described the first is not a 'birth fracture'. It is a badly treated tibial fracture in an infantile paralytic leg and is included only on account of its interest in simulating 'birth fracture'. Two bone-grafts had failed. One was an ordinary inlay, rather massive in size, and the other a sliding-down of the anterior half of the upper part of the tibia over the fracture. Both grafts were very large and were not ultimately successful (*see Fig.* 516). As will be seen from the radiograph (*see Fig.* 517), I shaved away the cortex on each side of the tibia above and below the fracture, took a big graft from the other tibia, split it, and placed one piece in each of the bared areas and tied them together with catgut. This was done very roughly and was quickly successful in obtaining union (*see Fig.* 518).

The second method consists in ignoring the zone of absorption and placing a strong bone-graft behind the tibia, implanting the graft into the back of the bone well above and below the site of non-union (*Fig.* 515). It is a sort of 'bony by-pass' and carries the weight directly from the upper part of the bone to the lower. The first patient (*Case* 2) in whom I performed it, had had innumerable operations—inlay grafts; sliding grafts; step-graft—all followed by absorption and cyst formation and persistent non-union. Some of these grafts had been performed before I saw him. There was complete non-union, and when he put his weight on the leg the fracture yielded like a joint, and the lower end of the upper fragment rested on the top of the foot. Incidentally the foot could be markedly dorsiflexed and calcaneus deformity gradually developed (*see Figs.* 519, 524, 526). It was while treating this boy that I began to feel that I was contending with an active process. Having successfully bridged gaps in bone, I excised the whole area of non-union and cyst formation, applied skeletal traction to overcome the acute angulation of the bones and shortening of the soft structures, and when the limb was straight I placed a graft across the gap. At first it seemed to progress satisfactorily, but as will be seen in *Fig.* 522 it was 'eaten into' at the level of the original fracture and the limb reverted to its old position. I then did the 'by-pass' graft I have described and the result was extremely satisfactory (*see Figs.* 523–525). Very cautiously as union proceeded protection was diminished until the boy was walking without any apparatus. Although he has of course considerable shortening and has to wear a thickened boot, he walks a long way to school and plays all games. He is an extremely rough and active boy and the leg has stood up to more than ordinary strain.

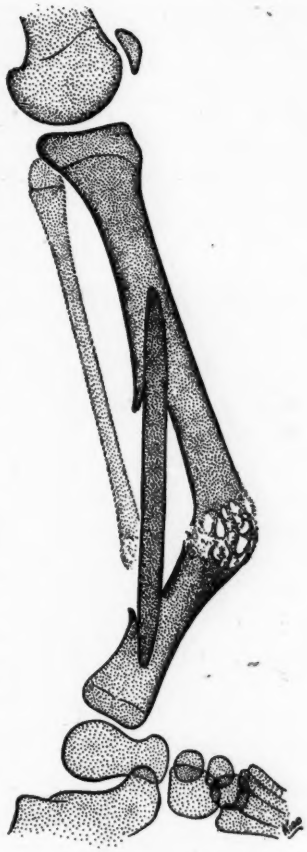

FIG. 515.—Sketch of 'by-pass' graft.

The third case, a little girl, was one year old when first seen. Deformity was slight and non-union was suspected but difficult to confirm. Complete and continuous immobilization was provided (not overlooking the factor of weight-bearing) for nine months, and still union was very unsatisfactory. The leg was opened,

non-union was demonstrated, and a small graft was introduced. Very careful immobilization was continued. Time went on, and after two and a half years' uninterrupted immobilization in plaster it became obvious that this fracture was going the way of other cases of congenital pseudarthrosis (*see Figs.* 526, 527). By this time the efficacy of the by-pass graft had become apparent. I inserted one in this child's tibia. By the same process of gradually diminishing protection she became free and has a strong leg and enjoys full activity (*see Figs.* 528, 529). Moreover, shortening has been prevented, and although the contour of the shin is not quite normal, this could be easily dealt with.

CASE REPORTS

Case 1.—H. W., boy, aged 11 years. Old-standing infantile paralysis left leg. Fracture, 1930. Bone-grafts left tibia, 1932–3 (*Fig.* 516). Developed some features similar to birth fracture: (1) Bone absorption and cyst formation; (2) Absorption of repeated massive grafts and resulting non-union in spite of full immobilization.

1936: Rough lateral grafts (*Fig.* 517).

Strong union 7 months later (*Fig.* 518).

FIG. 516.—*Case* 1, December 6, 1934. Non-union and absorption, in spite of (1) inlay, and later (2) sliding grafts and continuous immobilization.

FIG. 517.—*Case* 1 June 19, 1936. Lateral grafts in place (plaster).

FIG. 518.—*Case* 1, January 16, 1937. Solid bony continuity, seven months after operation.

Case 2.—N. K., boy. First seen, 1930, aged 3½ (*Fig.* 519). Step-graft and correction, followed by immobilization.

1933: Inlay graft.
1934: Sliding graft.
1935: Non-union (*Fig.* 520).
1935: Excision and traction and graft (two stages) (*Fig.* 521).
1936: Absorption recurred (*Fig.* 522).
July, 1936: 'By-pass' graft (*Fig.* 523).
February, 1937: Union (*Fig.* 524).
March, 1939: Solid bony continuity (*Fig.* 525).

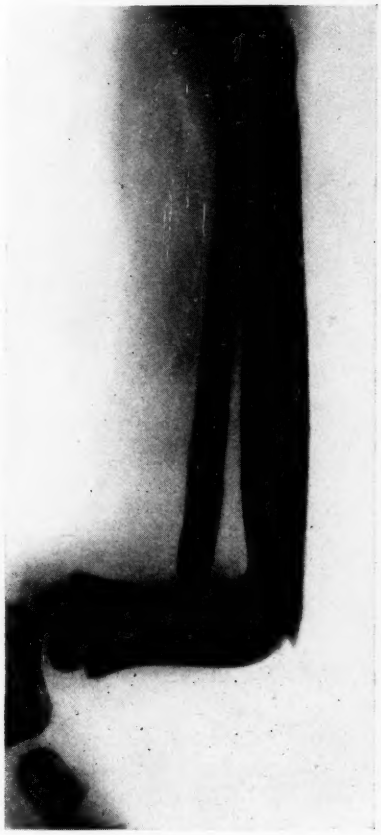

FIG. 519.—*Case* 2, October, 1930, 'birth' fracture. November, 1930, step graft and correction of deformity, followed by meticulous immobilization. (Note the calcaneus deformity.)

FIG. 520.—*Case* 2, 1935. Absorption and deformity worse, although strongly grafted in 1933 and carefully immobilized afterwards.

FIG. 521.—*Case* 2, 1935, after excision and traction: graft one month later.

FIG. 522.—*Case* 2, July, 1936, 'absorptive' process recurring.

FIG. 523.—*Case* 2, July, 1936, 'by-pass' bone-graft (in plaster).

FIG. 524.—*Case* 2, April, 1938, union of graft to shaft proceeding: also union of old fracture (note calcaneus deformity).

FIG. 525.—*Case* 2, March, 1939, solid bony continuity.

Case 3.—W. P., girl, aged 1 yr. 5 mth. First seen in 1932.
Immobilized in plaster-of-Paris for one year.
1933: Inlay graft.
1934: Recurrence of absorption (*Fig.* 526).
June, 1936: Non-union and increased deformity (*Fig.* 527).
October, 1936: 'By-pass' graft.
January, 1937: Union of graft and shaft commencing (*Fig.* 528).
March, 1938: Strong weight-bearing union.
January, 1939: Strong bony continuity. Fracture apparently also uniting (*Fig.* 529).

(Continuous immobilization in plaster-of-Paris 1932–7.)

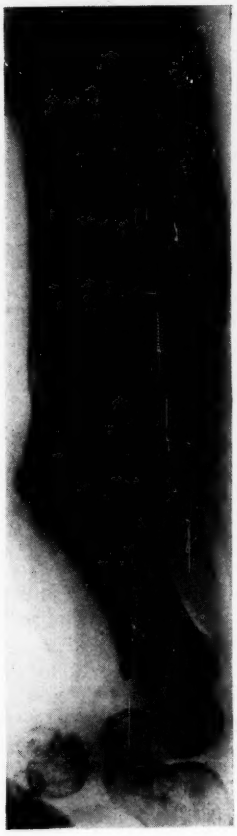

FIG. 526.—*Case* 3, December, 1934, absorption and deformity. One year after 'inlay' graft.

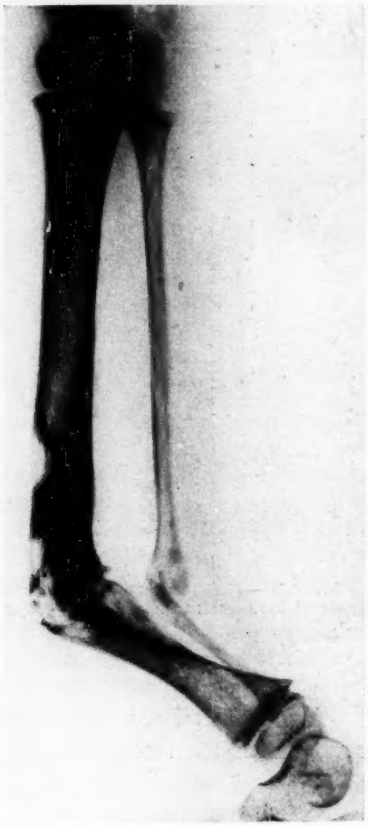

FIG. 527.—*Case* 3, June, 1936. Absorption and deformity increase, although never out of plaster-of-Paris since 1933.

FIG. 528.—*Case* 3, January, 1937, two months after 'by-pass' bone-graft which is commencing to unite to shaft.

FIG. 529.—*Case* 3, January, 1939, strong union of graft and shaft, and apparently union of old fracture.

SUMMARY

A way has been found and is described of obtaining in cases of congenital pseudarthrosis a strong, fully useful leg, of proper length.

SIALOANGIECTASIS

By GEORGE SWINBURNE
FROM THE DEPARTMENT OF PATHOLOGY, UNIVERSITY OF MELBOURNE

SIALOANGIECTASIS is a term which the writer suggests may be used to describe a condition of the salivary glands where the ducts and terminal ductules, and even the terminal alveoli, are dilated, resembling the dilatation of the bronchi and terminal bronchioles in bronchiectasis. The term is derived from the Greek σίαλον saliva + ἀγγεῖον vessel + ἔκτασις stretching out.

The term 'sialectasis' which has been used to describe the condition is not strictly correct etymologically, translating as 'a stretching out or dilatation of the saliva'. The term 'sialography', commonly used for the radiographic demonstration of the salivary channels, is open to the same etymological criticism. More correctly, it should be 'sialoangiography'.

CASE REPORT

J. M., male, aged 50 years.

The patient stated that in 1919, on his return from the war, he suffered an attack of what was apparently a left-sided acute suppurative parotitis. From the history, it would appear that pus formed and finally evacuated itself into the mouth, probably via Stenson's duct.

An inflammatory condition persisted, and at varying intervals during the next few years he had recurring acute inflammatory attacks in both parotid and both submaxillary glands. The patient could not remember the order of involvement of the respective glands. Various medicaments were injected into his parotid ducts from time to time, sometimes combined with dilatation, but without any improvement; the patient stated that his submaxillary glands were not treated.

Examination on February 11, 1938, revealed a slight enlargement of both parotid and submaxillary glands, but there was no definite lobulation such as Pyrah[6] described in his case. The ducts of the parotid and submaxillary glands, though dilated, were not palpable. The orifices of all four were larger than normal and slightly patulous, especially the parotid orifices, from which a viscid drop of secretion usually depended.

The epithelium of the cheek and tongue was normal and showed a normal degree of moisture. The submaxillary salivary glands were a little larger and firmer than these are normally, but there was no palpable enlargement of the cervical lymph-glands.

On one occasion in March, 1938, the patient was seen during an acute exacerbation in the left parotid. The gland was swollen and tender. By pressing from behind forwards over the parotid and submaxillary glands large quantities of mucopurulent saliva could be expelled. The accumulation was rapid, and such emptying could be performed at frequent intervals. It was his custom so to empty his glands regularly before each meal, and in addition this became necessary on occasion during the course of a meal. The saliva contained 3500 pus cells per cubic millimeter, and *Str. viridans* was obtained on culture. The smear showed the usual mouth organisms and that of Vincent's angina. There was no gross sepsis evident in connexion with his teeth, tonsils, or accessory nasal sinuses at the time of examination. No history of an attack of stomatitis, or of an operative procedure prior to the original attack, could be elicited.

X-ray examination showed no calculi in any of the glands. A sialogram made by injecting 1–2 c.c. of lipiodol into the duct of each gland showed gross dilatation apparently of all the ducts and alveoli, extending proportionately from the main duct along to the terminal

parts of the smaller ducts, producing by the filling of the distended terminal alveoli the appearance of a multitude of knobby extremities in the case of each gland. In the case of the parotid, sialograms showed both Stenson's ducts to be greatly dilated, but the left to a greater degree than the right (*Fig.* 530). Both Wharton's ducts were about equally dilated (*Fig.* 531), but the knobby dilatations in the body of the gland were larger than in the case of the parotid. This difference may be due to the lipiodol not penetrating to the terminal alveoli, and to a greater dilatation of the terminal ductules. The lipiodol penetrated particularly well in the case of the parotid glands; both injections were made under similar conditions, about 1½ c.c. of lipiodol being injected on each side in the case of the parotid gland, and injection stopped when the patient complained of a full feeling in the gland. This feeling was produced by the injection of slightly less lipiodol in the submaxillary glands.

In November, 1937, the patient was found to be suffering from pernicious anæmia. At that time he complained of weakness and pins and needles in his arms and legs for four months. He was short of breath on exertion and had lost 1½ stone in from four to six months.

Examination of his nervous system revealed a loss of epicritic sensation and diminution of protopathic sensation of glove and stocking distribution. Deep sensation, as evidenced

FIG. 530.—Sialogram of right parotid gland, showing the characteristic dilatations.

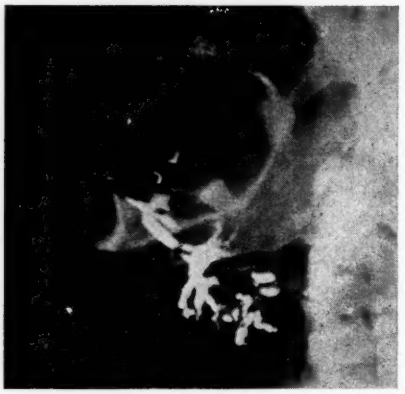

FIG. 531.—Sialogram of right submaxillary gland, showing a different arrangement of the dilatations.

by tests for joint sense, vibration sense, and discrimination between two points, was normal. The deep reflexes were diminished, but the plantars were flexor and the superficial abdominals present.

A fractional test-meal revealed a fasting content of 15 c.c., no bile, blood, or lactic acid, some increase in mucus, no free acid, and a very low total acidity.

A blood-count on December 20, 1937, showed 2,200,000 R.B.C., 5000 W.B.C., 53 per cent Hb, and a Colour Index of 1·2.

The average diameter of the red blood-corpuscles was 8·3 μ, there was anisocytosis and poikilocytosis, and a hypersegmentation of polymorph nuclei.

On January 7, 1938, the hæmoglobin was 69 per cent and the R.B.C. 2,900,000—that is to say, he was responding to treatment, which consisted of intramuscular injections of anahæmin (liver extract) 2 c.c. and Tab. Benerva (Roche) 1 t.d.s. The latter tablets contain vitamin B_1. His peripheral neuritis was thought to be due to a lack of vitamin B, not because of a lack in the diet, because the other members of his family were unaffected, but because of inadequate absorption in the intestinal tract owing to his pernicious anæmia. He continued to respond to treatment along these lines, with disappearance of his neuritis.

His mother died of "influenza", as did one brother and one sister. His father, two brothers, one sister, a wife, and one child were alive and well, and there was no history of pernicious anæmia in his family or apparent evidence of dietary insufficiency. His blood-pressure was 130/85 and the Wassermann reaction was negative.

DISCUSSION

It is generally considered that inflammatory conditions of the parotid glands are probably all due to ascending infections. The mouth is normally inhabited by a number of different organisms, and as long as the salivary glands function adequately, these are prevented from invading the glands by the flow of saliva and by the normal factors of cell resistance; but if there is any diminution of salivary activity as occurs in pyrexia, or after laparotomy, an ascending infection along the ducts often follows. Pavlov has experimentally proved that this diminution of salivary activity in pyrexia and after laparotomy is a constant phenomenon in dogs. In laparotomy cases the predisposition to ascending infection is greater in cases which are rectally fed; it is also greater in cases of gastric ulcer which are fed per rectum. However, in these cases it is nearly always only the parotid which is affected, and not the submaxillary glands. It has been suggested (Pyrah) that the mucous secretion of the submaxillary glands has a higher bactericidal property than the serous secretion of the parotid, but no experimental evidence was given in support of this statement. The infection of the parotid, if it occurs, usually appears about the end of the first week after operation or the onset of fever.

In 1931 Payne reported 4 cases of chronic parotitis in the BRITISH JOURNAL OF SURGERY, three arising from chronic infective foci in the mouth, and another in which chronic stomatitis had preceded the parotitis. In one of his cases there was a gross dilatation of both Stenson's ducts without any dilatation of the terminal alveoli, and because of its bilateral distribution he regarded the dilatation as probably congenital.

Barsony has reported a case with great dilatation of one Stenson's duct only.

Another case of Pyrah's gave a six months' history of a salty taste in the mouth and occasional swelling of the parotids, following a stomatitis. The sialogram showed a normal-sized Stenson's duct, but there were many small dilatations of terminal ductules, while others were normal. He regarded the sialogram of this case as depicting the earliest departure from normal.

The interest of the present case lies in: (*a*) The great dilatation of Stenson's and Wharton's ducts; (*b*) The dilatation of the small ductules or terminal alveoli; (*c*) The probable aetiology and pathology; (*d*) Relation to the development of pernicious anæmia.

In one reported instance where there was thought to be an obstruction near the orifice, the infection cleared up after the duct had been slit and lipiodol injected. There is no block at the salivary orifices in this patient, although there may have been originally before attempts at treatment. However, in this man the condition is so extensive, involving both parotid and both submaxillary glands, that a congenital dilatation with secondary infection has to be considered. There is, however, no evidence to support this hypothesis.

The question also arises in this patient as to why he developed pernicious anæmia. The salivary glandular abnormalities were present for years before its development, but may the appearance of the pernicious anæmia bear some relation to the salivary gland condition? This is not beyond the bounds of possibility, although there is again no direct evidence to support such a hypothesis.

SUMMARY

1. An unusually well-marked case of sialoangiectasis as a result of chronic parotitis is recorded.

2. The aetiology and pathology of this interesting condition is mentioned, and the findings compared with those of several previous observers. It is probable that the condition is secondary to inflammatory changes.

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POST-TRAUMATIC CEREBROSPINAL RHINORRHŒA WITH CASE REPORT

By WILLIAM GISSANE and B. K. RANK

In the medical writings of Bidloo[1]—about 1700—is recorded a case with escape of large amounts of fluid from the nose following a head injury; it had a fatal termination after eight months' duration. The escape of cerebrospinal fluid from the nose was first described in English literature in 1826 by Miller[2]; but it was not until 1899 that Sir St. Clair Thompson[3] first used the term 'cerebrospinal rhinorrhœa' in describing a case of this condition with unknown aetiology.

A study of reported cases of cerebrospinal rhinorrhœa would give the impression that it was most often spontaneous in origin, and that trauma was an uncommon cause. Sir St. Clair Thompson collected 21 cases up to 1899, only 1 of which had a history of previous head injury; a further 21 cases analysed by Johnston[4] up to 1926 included only 6 with a traumatic origin.

There are several known causes of a communication between the cerebrospinal fluid system and the nasal cavity. Anatomically it may exist via a bony defect in: (*a*) the walls of the frontal sinus, (*b*) the cribriform plate of the ethmoid, or rarely (*c*) the sphenoidal sinus. Such bony defects may be caused by trauma with fracture, by damage at an operative procedure, by pressure necrosis or erosion of a tumour, by hydrocephalus, or it may primarily exist as a congenital defect.

Despite the evidence of the literature, fracture by trauma is probably the commonest cause of such a bony defect, and to establish the necessary fistulous communication between the subarachnoid space and the lining membrane of the affected sinus or nasal cavity a comminuted fracture with displacement and tearing of neighbouring soft tissues is the most dangerous type. Such fractures are most commonly seen nowadays among air pilots, as the upper part of their face makes violent contact with the instrument panel in air crashes. McIndoe[5] says that comminuted fractures of the floor of the anterior cerebral fossa, complicated by persistent cerebrospinal rhinorrhœa, are not uncommon injuries to air pilots.

On available information it is impossible to determine the incidence of this condition as a complication of fractured skull. We assume that with increased numbers of skull and upper facial fractures it must become a more common clinical entity. Julian Taylor[6] has said "it is one of the most serious complications of cranial injury".

TYPES OF POST-TRAUMATIC CEREBROSPINAL RHINORRHOEA

Clinically and radiologically two main sites of fistula through a fracture line might be distinguished: (1) A fracture involving the cribriform plates of the ethmoid; (2) A fracture involving the roof of the frontal sinus.

Though often difficult, precise radiological examination may well distinguish these two types. Such evidence may be confirmed by clinical examination of the

nasal cavity. In the former the cerebrospinal fluid will be seen coming from the roof of the nose; in the latter, when the fistulous communication is via the frontal sinus, the fluid loss is from between the middle and inferior turbinates. It must be recognized that a fracture line may involve both these regions.

The practical value of this distinction lies in the surgical approach to the fistula. The difficulties of approach to the roof of the frontal sinus are not great; those to the cribriform plate are better left in the hands of a specialist in cerebral surgery.

Of more importance concerning prognosis and treatment, is the distinction as given by Cairns[7] of (1) those cases of cerebrospinal rhinorrhœa occurring in the early or acute stage of head injury, and (2) those occurring as a delayed complication of head injury.

1. Early Post-traumatic Cerebrospinal Rhinorrhoea.—The loss of excess of blood-stained fluid from the nose following the more severe types of fracture of the anterior cerebral fossa is not a very uncommon clinical observation. These are, in every sense of the term, open fractures, and, following first principles in treatment of open fractures, one might consider early operation to cleanse the wound and close the opening. Cairns[7] has advised early interference in such cases if known infection of the nasal sinuses is present, and Coleman[8] advises active interference in depressed fracture involving the frontal sinus.

Among those cases which survive the initial head injury, we are familiar with the great tendency of such fistulæ to heal spontaneously in a very short time, with complete cessation of cerebrospinal fluid loss. During the period of natural healing it is our custom to leave the nasal passages severely alone, as any active interference by nasal douches of saline or antiseptics carries more dangers than benefits, a plug to the nasal cavity being especially dangerous.

In those fractures associated with gross deformity of the upper facial bones, we believe that the facial fractures should be reduced and held by any means other than intranasal splintage, and the fistula left undisturbed to heal by natural processes. If the fracture has obliterated the nasal passages the method (advocated by McIndoe) of re-establishing the patency of these passages by introducing rubber drainage tubes not only recommends itself as the proper emergency treatment of such a serious facial disfigurement, but also allows the necessary free drainage of cerebrospinal fluid loss, any intranasal blockage of which may well increase the risk of meningeal infection.

2. Delayed Post-traumatic Cerebrospinal Rhinorrhoea.—This is the group of surgical importance. The condition is usually first noticed when the patient is up and about after recovery from the initial cerebral injury.

The recorded pathology in those cases operated on, as in that here reported, indicates that the requisites for spontaneous healing are absent. Adhesions are found about the persistent bony defect, through which a fistulous track is permanently established between the subarachnoid space and the cavity of the nose or nasal sinus. Through the fistula leakage of cerebrospinal fluid occurs at a rate dependent on the size of the fistula and the relation between cerebrospinal and intranasal pressures. This relationship may be reversed during sneezing or nose blowing, with an influx of air from the nasal cavity—hence the almost invariable association of cerebrospinal rhinorrhœa with varying degrees of intracranial pneumatocele. As long as the fistula persists there is ever present the danger of influx

of micro-organisms from the nose causing fatal infective intracranial complications. A review of the literature leaves no doubt as to the usual fatal termination of these cases if the fistula is allowed to persist.

TREATMENT OF DELAYED POST-TRAUMATIC CEREBROSPINAL RHINORRHOEA

Sir Arnold Lawson[9] stated that this condition is always fatal in the course of time from secondary meningitis unless the defect is closed by natural healing processes or by operative interference. This view is substantiated by a study of the recorded cases. Spontaneous healing is unlikely in an established fistula. Though the operative mortality is high (30 per cent) it is not as high as the death-rate from septic complications among cases not subjected to operation.

There is no established method of operative closure of the fistula or of obliteration of the bony defect. Many methods have been used: Excision of the track and direct suture of the dural defect[7]; occlusion of the region between dura and skull by grafts of pericranial tissue,[4] fascia lata,[7] or muscle[8]; obliteration of the fracture line by Horsley's wax.[10] The high operative mortality has been advanced as a reason for not operating on these patients.[11] Sepsis following operation is the chief cause of this high mortality. This high incidence of infection might well be reduced if an effective method of occlusion of the fistula could be established by using a technique requiring no buried suture material and a graft of high viability to cover the bony defect.

We here report a case of delayed post-traumatic cerebrospinal rhinorrhœa in which a successful result was obtained by operative closure of the defect.

CASE REPORT

J. G., male, aged 16, was admitted to St. James' Hospital, having been found two hours previously in a garden below the window of his bedroom on the fourth floor. He was unconscious, and the facial abrasions and bruising indicated that the main force of his fall had been received on the region of his forehead. There were no other injuries and no obvious signs of a localized cerebral lesion.

Next morning he had recovered consciousness. There was marked periorbital hæmatoma, subconjunctival hæmorrhage, and chemosis of the left eye. He could give no history of the event and had retrograde amnesia of several hours. X-ray examination revealed a fracture of the left frontal bone (*Fig.* 532).

Apart from persistent and severe left frontal headache, the clinical course was uneventful until the sixth day, when headache became occipital and very intense. Temperature rose to 104°. There was marked neck stiffness and other signs of severe meningeal irritation, and the patient rapidly became comatose. Lumbar puncture produced turbid blood-stained cerebrospinal fluid under increased pressure—it contained 150 mg. of protein per 100 c.c. and numerous polymorphonuclear cells. At no time were organisms isolated from the cerebrospinal fluid. After three days with twice-daily lumbar puncture and administration of M & B 693 (15 gr. four-hourly) the temperature and signs rapidly resolved, the patient regained consciousness, and the lumbar fluid cleared.

He was discharged to a convalescent hospital ten weeks after his accident, still complaining of recurrent frontal headaches, but otherwise well.

Four weeks later (fourteen weeks after his accident) he was re-admitted to St. James' Hospital, still with headache and now complaining of diplopia looking to the right. He also complained, for the first time, of a "dripping of salt-tasting fluid from his left nostril". This he had noticed since he had been up and about, approximately ten weeks after his accident. The flow was intermittent and always worse after regaining the erect position

if he had been stooping. On these occasions he noticed a "noise in his head like that of the last drop of water running out of a bath". There was no definite relation between intensity of headache and the flow of fluid from his nose.

FIG. 532.—Lateral radiograph of skull taken on day after the original injury, showing intercranial pneumatocele already present.

The diagnosis of cerebrospinal rhinorrhœa was confirmed by chemical and pathological examination of the fluid. About 10 oz. of fluid, macroscopically clear, could be collected in twenty-four hours. It contained 200 mg. of protein per 100 c.c. and 80 cells per c.mm. (90 per cent polymorphonuclears). *M. catarrhalis*, *Staph. albus*, and diphtheroid organisms were cultured from the fluid on two occasions. At this time the lumbar fluid at 150 mm. pressure was clear and sterile; protein 40 mg. per 100 c.c. and cell content normal.

FIG. 533.—Oblique radiograph, showing the fracture line involving the roof of the frontal sinus.

At this stage more precise radiological examination revealed the fracture of the left frontal bone involving the roof of the frontal sinus (*Fig.* 533). Rhinological examination revealed "a normal healthy nose and no evidence of infection of the accessory nasal sinuses".

After three weeks' observation it was noted that the amount of cerebrospinal fluid loss from the nose was increasing, and it was decided to attempt occlusion of the fistula.

TECHNIQUE OF OPERATION (performed four months after his original injury).—Under general anæsthesia (endotracheal technique) with field block supplement, a long frontal scalp flap was reflected from just within the hair margin, giving a bony exposure down to the line of the supra-orbital ridge. The vertical fracture line was observed on the left frontal bone

down to the region of the left frontal sinus. A left frontal osteoplastic flap including the fracture line was reflected, exposing the dura of the left frontal pole (*Fig.* 534). The high intradural tension was reduced by intravenous hypertonic saline. This allowed sufficient retraction of the frontal pole to display the roof of the frontal sinus. The dura was found to be closely adherent along the fracture line. Separation of these adhesions revealed about the middle of the roof of the sinus a bony defect approximately 0·5 cm. wide. Through this defect were two fistulous tracks communicating with the mucosa lining the frontal sinus. These were diathermized. Behind the defect were more soft adhesions, easily broken by blunt dissection. Elsewhere the dura was apparently normal.

The bony defect was now covered by a free periosteal graft taken from the tibia. The graft, 2 in. × 1 in., was placed along the fracture line with the rough surface toward the

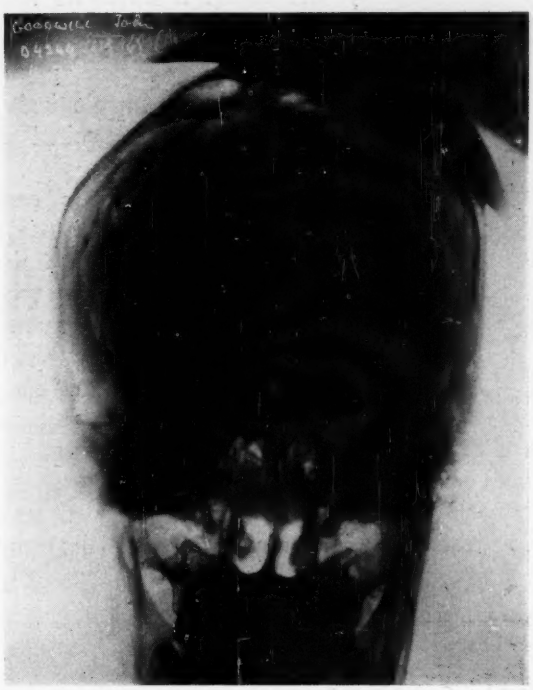

FIG. 534.—Post-operative radiograph, showing the surgical approach used.

frontal sinus. Release of the frontal lobe gave the necessary even pressure to keep the graft in position—there was no necessity to suture it in place. The bone-flap was replaced and the wound closed.

SUBSEQUENT PROGRESS.—Convalescence was afebrile throughout and uneventful. The wound healed by first intention and the patient was up and about three and a half weeks after operation.

Since operation (June 16, 1939) there has been no recurrence of cerebrospinal rhinorrhœa. Headache is infrequent and mild. His only complaint is persistence of diplopia looking to the right.

Comment.—In its cause, its delayed onset, and the clinical course this case presents similar features to others described.

Special features of interest are :—

1. The severe meningitis which complicated the original injury may have been of an aseptic reactionary type, but was most probably infective in origin and an index of communication with the nasal cavity. The response to M & B 693 is noteworthy.

2. The points of note in the operative technique are the use of a diathermy knife in excision of the fistula, obviating the need for suture material, and the use of periosteum as a graft. Periosteum was chosen because it is a vascular fibrous tissue of high viability accustomed to a bony nidus, and its pliability allows close adaption to the surface contours about the fracture defect.

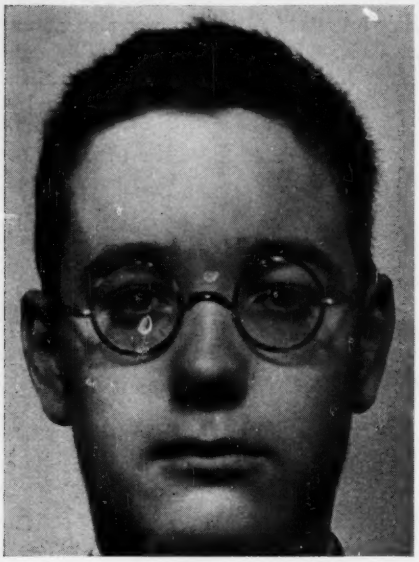

FIG. 535.—Photograph taken two months after operation.

The surgical approach used gave good access and it leaves no visible scar (*Fig.* 535).

SUMMARY

1. The histological and aetiological aspects of cerebrospinal rhinorrhœa are discussed.

2. Post-traumatic cerebrospinal rhinorrhœa is discussed from a clinical standpoint. The importance of Cairn's distinction of early and delayed types is emphasized regarding the management of this condition.

3. The tendency for spontaneous healing is recognized in the 'early' type, and conservative treatment recommended.

4. The dangers of conservative treatment of the 'delayed' type are emphasized and operative treatment is advocated.

5. A case of the delayed type is reported, with a successful result obtained by operation, using a graft of periosteum. The special features of the case are discussed.

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ON THE MUSCLE-FIBRES OF THE ANAL SUBMUCOSA, WITH SPECIAL REFERENCE TO THE PECTEN BAND

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INTRODUCTION

THE findings described below are the outcome of an investigation into the nature of the pecten band, a thickening of the anal submucosa commonly regarded as a pathological entity and stated to be associated with chronic inflammatory or circulatory disturbances.

The pecten band was first noted by Miles (1919), who described it as "a circular band varying in thickness and density and giving the impression to the examining finger as if a rubber umbrella ring had been inserted beneath the skin at the anal margin". On purely theoretical grounds he concluded that it was composed of fibrous tissue and was invariably pathological in origin; however, no histological studies were instanced to support this view.

The existence of this band, as well as the favourable results obtained by its division ('pectenotomy'), in a variety of pathological conditions of the anus was confirmed by Abel (1932), whose views as to the nature of the pecten band were identical with those of Miles, though some confirmatory histological evidence was offered in his case in the form of an unstated number of biopsy examinations. In concluding that the pecten band was of fibrous nature both Miles and Abel appear to have been influenced by its physical characters, and contrast the white fibres composing it with the red fibres of the external sphincter contiguous with it. This pallor, however, though incompatible with striated muscle, is quite compatible with unstriated muscle (Cajal, 1933).

J. W. Morgan (1934) found pectenotomy a curative measure in many pathological conditions of the anus: he submitted 7 biopsy specimens of the pecten band to histological examination and found fibrous tissue in 5 and unstriated muscle in 2. Spieseman (1939) commented favourably on the results of 250 cases of pectenotomy, including 48 cases operated on for intractable pruritus ani: he made the interesting admission that he only became aware of the existence of the pecten band after reading the work of Miles.

It would seem a matter of surprise, in view of the findings of the above workers, that universal recognition has not been accorded by proctologists to the existence of the pecten band, let alone the benefits derived from its division. Thus Gabriel (1937) in his text-book of rectal surgery omits all reference to the subject.

In view of the paucity of histological evidence submitted by writers on the pecten band, it was decided to examine a number of post-mortem ano-rectal specimens as well as some biopsy specimens of the pecten band. In all, 7 ano-rectal and 4 biopsy specimens were examined.

INVESTIGATION

Section through the Ano-rectal Junction.—Five of the post-mortem specimens were from cases of carcinoma of the rectum, and were chosen because of pronounced thickening of the submucosa: the specimens themselves were not involved in the malignant change. The remaining 2 were from male cases, aged

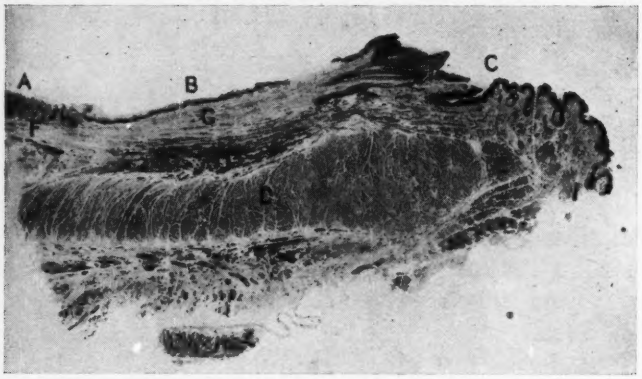

FIG. 536.—Ano-rectal junction. A, Columnar epithelium of rectum; B, Anal epithelium; C, Epidermis; D, Internal sphincter; E, External sphincter; F, Muscularis mucosæ; G, Superficial part of anal submucosa, with few muscle-fibres; H, Compact muscle-bundle in deep part of anal submucosa. (× 2·8.)

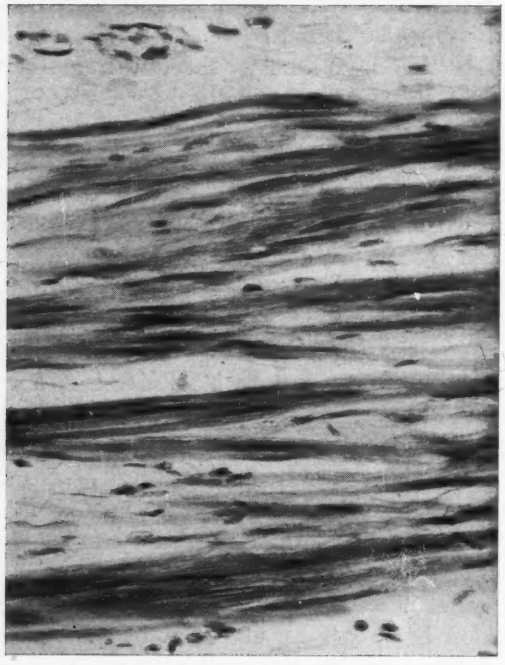

FIG. 537.—Muscle-fibres of anal submucosa. (× 240.)

60 and 30 respectively, not presenting any local pathological changes. They were chosen because they presented no perceptible thickening of the submucosa.

As a result of histological examination the following points emerged:—

1. All the specimens showed the presence of bands of involuntary muscle-fibres in the submucosa.

Fig. 536 is a low-power microphotograph of a section, showing a well-developed muscle-band in this region; *Fig.* 537 a high-power microphotograph demonstrating cytologically the muscle-fibres composing this band. It can be seen that this muscle-band is most compact in the deepest part of the submucosa, where it is in close contact with the internal sphincter: it appears to be quite distinct from the latter, however.

The more superficial part of the submucosa also contains muscle-fibres, less clearly shown in *Fig.* 536: the interval between them, filled by fibrous tissue, increases as the epithelium is reached.

2. All specimens showed a continuity between the muscularis mucosæ of the rectum and the superficial fibres of the submucosa muscle, the latter terminating at the level of commencement of the true epidermis.

3. The specimens differed considerably among themselves, both as regards thickness of the submucosa and the presence of a compact muscle-band. This is set out in the following descriptive table :—

No. of Specimen	Description of Case	Thickness of Submucosa	Compact Muscle-band easily seen with Naked Eye	Muscle-bundles Detected with Lens
A1	Rectal carcinoma	4 mm.	+	+
A2	Rectal carcinoma	1–3 mm.		+
A3	Rectal carcinoma	2 mm.		+
A4	Rectal carcinoma	2–3 mm.		+
A5	Rectal carcinoma	3 mm.	+	+
A6	Male, aged 60	2 mm.	+	+
A7	Male, aged 30	2 mm.		+

From naked-eye appearance alone it would be easy to understand how *Cases* A1, A5, and A6 might present clinically palpable bands (' pecten bands ').

4. The extent of submucosa and muscle present depends to some extent on the region selected for cutting. This was clearly seen when comparing two sections taken from different parts of the ano-rectal specimen as was done with A6 and A7.

5. None of the specimens showed evidence of chronic inflammatory change in the anal epithelium or its submucosa, such as round-celled infiltration.

Biopsy Specimens of Pecten Band.—

1. All the specimens examined contained involuntary muscle-fibres.

2. Some consisted of compact muscle-bundles with hardly any fibrous tissue; others contained an equal amount or an excess of fibrous tissue.

3. It was demonstrated that the proportion of muscle present depended on the situation of the tissue cut. This was done by dividing one of the specimens into five fragments: one of them was found to consist entirely of muscle-fibres; the others, which were all partly lined by anal epithelium, contained muscle-fibres to a varying degree, there being few near the epithelium, but an increasing number at the deeper levels.

A reconstruction of these fragments almost inevitably led to a picture of the fibromuscular submucosa as seen in *Fig.* 536.

4. None of the specimens showed evidence of chronic inflammatory change.

DISCUSSION

Histology of Normal Anal Submucosa.—The pathological significance, if any, of the appearances described can only be determined with reference to the normal histology of the ano-rectal junction. Histological literature, however, is scanty in its references to this region. The considerable text-books of Jordan (1934), Bailey (1936), and Maximov and Bloom (1938) do not deal with this region at all. Schafer (1938) describes the anal canal in some detail, even mentioning the anal glands opening on the mucous membrane, but does not refer to muscle in the submucosa. Piersol (1929), Piette (1931), and Bremer (1936) agree as to the occurrence of muscle-bundles in the rectal columns of the anal canal, Bremer stating that these bundles represent the termination of the muscularis mucosæ. In this Branca and Verne (1934) concur.

In view of this conspiracy of silence, the question arises whether the muscle-fibres observed in the submucosa are peculiar to the specimens examined, or whether they are normal histological constituents, hitherto overlooked by histologists.* On this question J. W. Morgan (1934) makes the illuminating comment that " the current descriptions of the anatomy of the anal canal found in modern text-books of anatomy and proctology are frequently incorrect. As often happens, pathological investigations lead to closer examination of anatomical detail and bring to light points which traditional descriptions have neglected, or but cursorily mentioned." That traditional descriptions should have neglected such striking appearances as are shown in *Fig.* 536 can only be due to the absence hitherto of pathological investigations leading to a closer examination of histological detail of the region concerned.

It would seem difficult to imagine how any pathological condition could give rise to new muscle-fibres in the anal submucosa, and the conclusion seems inevitable that these fibres are normal constituents, varying in amount in different subjects and sometimes manifesting themselves clinically as palpable bands whose nature is readily demonstrated on biopsy.

It is curious that the only confirmation in the literature of the occurrence of muscle-fibres in the anal submucosa was found in McCallum's *Text-Book of Pathology* (Fig. 257), a section of the ano-rectal junction illustrating thrombosis in hæmorrhoids, distinctly showing muscle-bundles underlying the stratified squamous epithelium of the anal canal, though the text contains no reference to this naturally irrelevant feature.

The Histology of the Pecten Band : Discussion of Conflicting Evidence.—It is necessary to reconcile, if possible, our own findings—clearly indicating the predominance of muscle in the biopsy specimens—with those of Abel, who states that fibrous tissue only was found, and of J. W. Morgan, who found muscle-fibres in 2 only out of 7 specimens. Such reconciliation should not prove difficult if it is borne in mind that the muscle-fibres predominate only at the deeper levels of the submucosa, and if sections are cut at a more superficial level muscle-fibres might be so few as to be regarded as possessing no significance, or even to be overlooked.

The Pecten Band as a Normal Structure.—While Miles asserts that the pecten band does not occur in the healthy anal canal, both Abel and Spieseman agree that occasionally a pecten band may exist without any associated pathology in the ano-rectum.

* An account of the literature would be incomplete without referring to the detailed study of the surgical anatomy of the anal canal by Milligan and C. Naunton Morgan (1934). That the myologically unimportant region here discussed was simply overlooked is the frank explanation made personally by Mr. Morgan to one of the authors (J.F.) for the absence of reference to muscle-fibres in the submucosa.

It is clear that even if a pathological condition is coexistent with a pecten band, this association may be incidental rather than aetiological. J. W. Morgan accepts this possibility, though he considers that in most cases of such association the pecten band is the primary factor in producing the lesion.

The Pecten Band as a Pathological Structure.—The therapeutic effects so persistently claimed by proctologists following the division of the pecten band, particularly in the series of cases of pruritus ani treated by Spieseman, strongly suggests that this structure does play some part in producing or maintaining such anal lesions as are associated with narrowing of the anal lumen.

The role which the pecten band in such a case must play would not be that of a fibrous band, as hitherto maintained, but that of a muscle band producing its effects through either: (*a*) Spasmodic contraction from a local irritation (e.g., fissure); or (*b*) Spasmodic contraction from functional causes, either central or local (e.g., pruritus); or (*c*) Hypertrophy, resulting from chronic spasm or other cause, and leading to simple stenosis (e.g., some forms of constipation).

Muscularis Submucosæ Ani.—It is proposed to apply this term to the muscle layer demonstrated in the anal submucosa, its superficial fibres only being continuous with the muscularis mucosæ of the rectum.

SUMMARY

1. Seven post-mortem specimens of the ano-rectal junction were examined histologically: in all these the submucosa showed the presence of involuntary muscle-fibres, in some cases forming a compact band close to, but distinct from, the internal sphincter.

2. The superficial bundles of the above muscle-fibres are directly continuous with the muscularis mucosæ of the rectum.

3. Four biopsy specimens obtained from patients with an annular thickening of the anal submucosa ('pecten band') showed this thickening to contain dense bands of muscle-fibres. It is concluded that the pecten band is a palpable manifestation of a well-developed submucosal muscle.

4. The role which the muscular pecten band might play in the pathology of anal conditions is indicated.

5. The term 'muscularis submucosæ ani' is proposed for the submucosal muscle layer, which has hitherto not been described.

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A CASE OF ADRENAL CORTICAL HYPERPLASIA, ASSOCIATED WITH PSEUDO-HERMAPHRODITISM

By I. DOUGLAS MILLER and P. J. KENNY

HISTORY

The patient was to outward appearance a male, and was 10 years old. The child when born was a healthy baby weighing 9 lb. It is stated that the accoucheur made an observation that the child was a hermaphrodite, but on what evidence this was based is not known. There was no increase in weight, and no visible growth until the tenth month of life, when the infant began to grow. At the age of 1 year he weighed 10 lb. Normal growth continued until the age of $2\frac{1}{2}$ years (*Fig.* 538),

Fig. 538.—Child aged 2.

Fig. 539.—Child aged 4.

when a sudden increase occurred, so that he was over 4 ft. high at 4 years of age (*Fig.* 539), and at the age of 6 he was over 5 ft. high. Since then, growth, if any, had been very slight, and he had not grown during the last year. Pubic and axillary hair appeared at 4 years, and at this time his external genitals began to develop, rapidly becoming adult in size. He had well-developed pubic hair and a man's voice. He had had to shave since the age of 7 years.

At the age of 4 years he was taken to a physician on account of blood-stained motions, for which no cause could be found. A note was made at this time that he was obviously not a hermaphrodite, but that he had a moderate degree of

hypospadias, with a penis curved like a bow with convexity upwards. Some months later the penis was freed, but no attempt to cure the hypospadias was considered advisable at this stage.

The opinion at this time was that his condition was one of pineal tumour, and as radiography of his skull revealed a rather wide sella, he was given pituitary gland substance for six months.

At the age of $4\frac{1}{2}$ he began to get attacks like appendicitis, and at this time X rays revealed some asymmetry of the sacrum and premature ossification of the bones.

Since the age of 5 years he had been very attracted to adult women, and showed great jealousy of anyone coming into contact with the objects of his affection. He used to have an uncontrollable temper over such matters, and for this reason could never be allowed to leave home.

His mother could not send him to school or allow him any social intercourse, and had to employ a governess, a source of great trouble on account of his jealousy over her.

He was never a child in his amusements and never played with toys. He would play cricket with other boys, but would quickly lose patience with them on account of his more mature outlook. He liked trains and enjoyed going round his father's farm.

He was formerly very energetic, but lately had been very studious and only with difficulty could he be got away from his books.

During the last three months he had been getting attacks of severe epigastric pain almost daily, but on several occasions had intermissions of three weeks' freedom. These attacks were not accompanied by vomiting, but had latterly been associated with nausea. He had often complained of a pain in the left side.

His skin was dark at birth, and had got slightly darker.

During his stay in hospital his behaviour was very good. He was most tractable and was generally well liked. He read most of the time—mostly adventure books. He was not at all troublesome to nurses, but did display a distinct preference for one nurse, whom he liked always to attend to him and for whom he would do what was required.

Family History.—There was one stillborn brother with hypospadias. Another brother is normal.

FIG. 540.—Child aged 10, at the time of investigation.

PHYSICAL EXAMINATION

The patient was to all appearance a male, with the facies of a youth of 18 years, a fine hair on his cheeks, upper lip, and chin (*Fig.* 540). There was a slight acneiform eruption on the forehead. The complexion was olive, more so on his abdomen than elsewhere. The breast areolæ were very dark. His weight was 8 st. 3 lb., his height just over 5 ft. His frame was well covered, but not obese. The limbs were stocky and muscular, but the hands were chubby like those of a child. Pubic and axillary hair were very thick. The external genitals were well developed, but there was complete

cryptorchidism, and perineal hypospadias. The teeth were a complete adult dentition, with the exception of the 3rd molars. His voice was that of a man.

Examination of the abdomen revealed the left epigastric region and hypochondrium to be fuller than the right. There was a sensation of a mass descending out of the right hypochondrium on deep inspiration.

The right kidney was readily palpable. An intravenous pyelogram showed both kidneys well. The upper calices of the right kidney were irregular, but this was considered to be due to spasm.

Perirenal insufflation of oxygen was done: 200 c.c. were injected into the left side and a radiograph revealed the outline of the kidney and suprarenal to be normal. An attempt was made to obtain a picture of the right side, but the colon obscured the shadows. There was a suggestion of a fairly large shadow surrounded by air. Air passed across the midline and showed a clear area in the corresponding paravertebral area on the other side. Several perirenal injections of oxygen were performed, but none gave a satisfactory picture, and the outline obtained on the left side was actually misleading. We are not impressed by the value of this method.

OPERATION

It was decided to expose both suprarenals from the back in the method advocated by Young. The right side was done first, by means of a high lumbar kidney exposure. No adrenal tissue could be found, though there was a most satisfactory exposure of the field. The problem then presented as to whether it

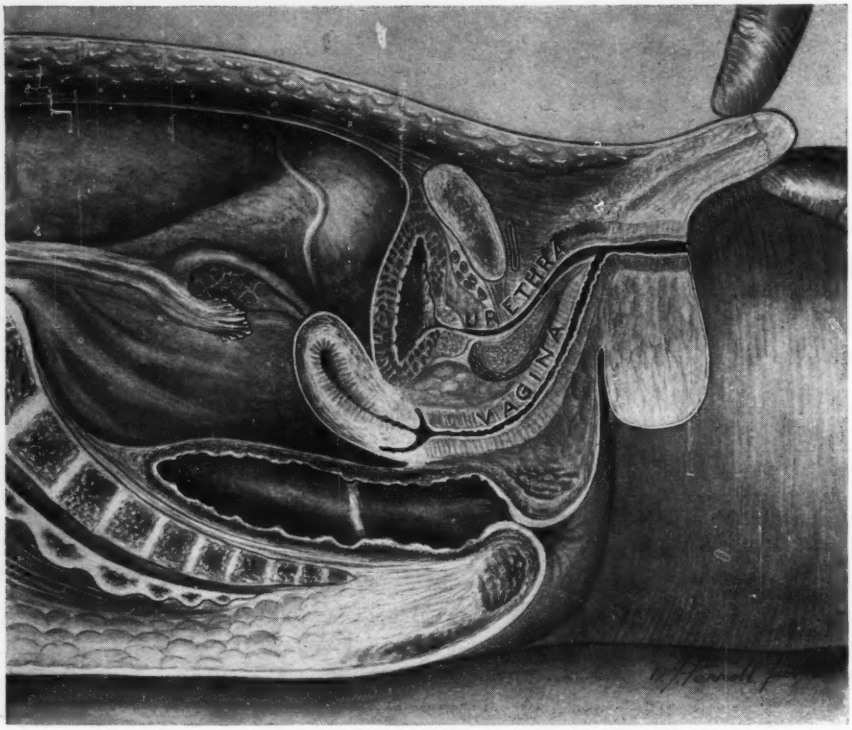

FIG. 541.—Sagi al section, showing vagina opening into urethra, just above the perineal hypospadias.

would be wise to expose the left side in view of the fact that any tumour or adrenal there could not be removed in the absence of adrenal tissue on the right side. It was decided to explore the abdomen. Complete female organs were found in the pelvis, consisting of a very small uterus with two normal tubes and two small ovaries (*Fig.* 541). The left suprarenal was palpated and found to be from three to four times the normal size, but of normal consistency and homogeneity. The pelvic organs were removed, and a small vagina, which was just large enough to admit a No. 12 rubber catheter, was found, and communicated with the urethra, which opened in the perineum (*Fig.* 542). A subacutely inflammed appendix was removed, which would probably have accounted for his abdominal pain, and the abdomen closed.

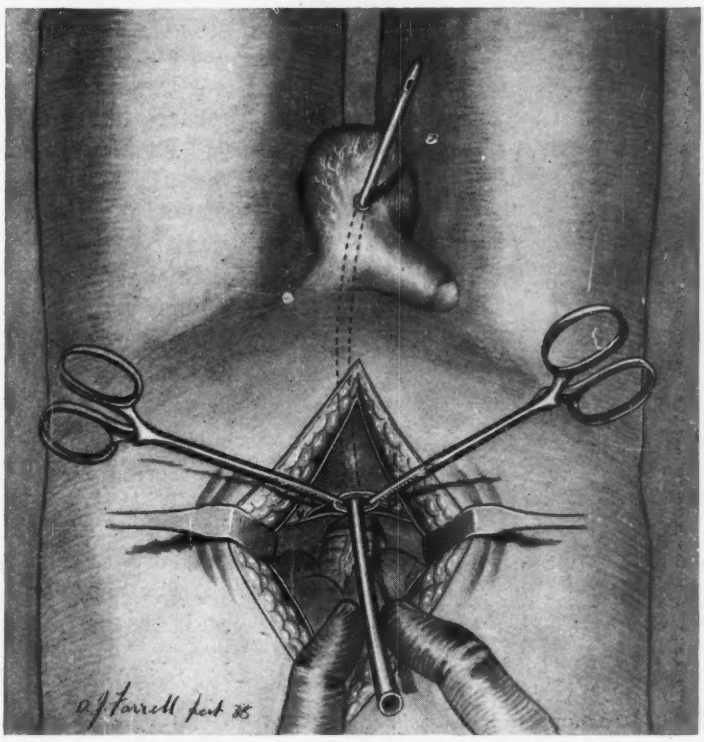

FIG. 542.—Drawing of parts, after removal of the uterus and its appendages. A catheter passing through the vagina emerges from the perineal hypospadic opening of the urethra.

His reactions under anæsthesia were very strange, as throughout the long operation his blood-pressure consistently rose to about 170 mm. from a pre-operative level of 80 mm.

After operation the reverse was found, and next day he was profoundly collapsed, and despite stimulant treatment and the intravenous administration of fluid and blood, he died about thirty-six hours after operation.

POST-MORTEM FINDINGS

Death had apparently been produced by an intraperitoneal hæmorrhage, as blood-serum was found in the peritoneal cavity and blood-clot was present in abundance in the right iliac fossa, extending down into the pelvis.

The suprarenal mass and the pineal and pituitary glands were removed for examination. There was no evidence of any male gonad. The left suprarenal was from six to eight times the normal size, but quite homogeneous and of normal consistence.

PATHOLOGICAL REPORT

(MR. A. H. TEBBUTT)

The specimens received were the left adrenal gland, the uterus and adnexa, and the pituitary gland (autopsy specimens).

1. *The Left Adrenal* (*Fig.* 543).—This was grossly enlarged and weighed 40 g. It was only in a fair state of preservation. The enlargement was due to cortical hyperplasia and there were no nodules or adenomata.

FIG. 543.—Left suprarenal. (*Actual size.*)

Paraffin sections stained with hæmatoxylin and eosin show a characteristic zona glomerulosa of normal dimensions, inside which is a greatly widened fasciculate and reticular zone, not well demarcated, the predominant cell of which is a large oval or round cell with small deeply staining nucleus and much eosinophilic cytoplasm (*Fig.* 544). In some areas blurring of structure is obvious, due to post-mortem changes, and nothing can be said of the medulla for this reason.

Paraffin sections were treated by bichromate and stained by ponceau-fuchsin after the method of Vines,[1] using ponceau 2 R obtained from Gurr (London). Practically all the cortical cells stained brilliantly as illustrated by Vines, and in considerable contrast to the adrenal of a woman in the fourth decennium dying about this time. But subsequent employment of this stain for a variety of tissues including the adrenal seems to show marked affinity of this stain for cytoplasmic structures. I am not aware of any error in technique. The modification of the stain since described[2] has not been used. All that I feel justified in saying, therefore, is that the hyperplasia of the cortex in this case is very significant.

2. *The Uterus and Adnexa.*—The uterus is somewhat small, the Fallopian tubes well developed, and the ovaries not greatly reduced in size—one 2·7 cm. long and 7 cm. greatest thickness, the other 2·0 cm. long by 0·6 cm.

Sections of the uterus show a thin layer of endometrium with glands in the quiescent phase and a stroma less cellular than normal.

Bisection of the ovaries shows visible follicular cysts. The surface is smooth and shows no evidence of follicles or corpora. Microscopical examination reveals numerous primordial ova in the cortex and a few Graafian follicles. One large follicle with ovum in situ shows a definite theca interna in which are a few large cells with clear cytoplasms. The naked-eye cysts have a single or several layers of small cubical cells and are considered to be atretic follicles.

There is a well-marked vascular hilum in which are cross-sections of tubules lined by columnar epithelium. These are interpreted as persisting junctional tubules. One would expect some œstrin production from such ovaries.

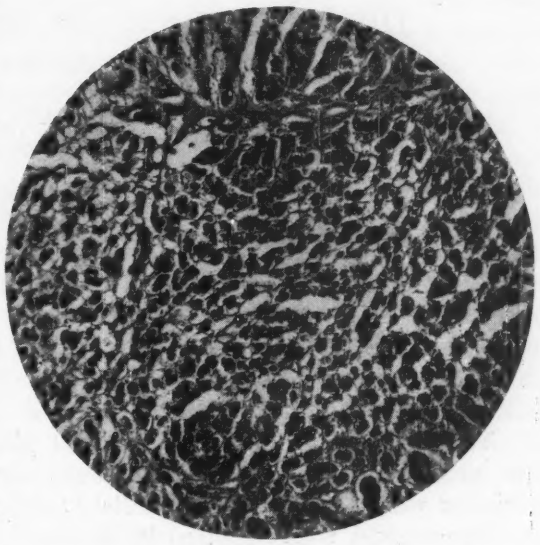

FIG. 544.—Section of suprarenal (*see* text).

3. *The Pituitary Gland.*—This appeared to be of about normal size. All cell types are present in the pars anterior. The esinophil cells have a small deeply stained nucleus and much cytoplasm and are numerous in all parts, though greatly predominating over other types in some areas.

COMMENT

A remarkable instance of male pseudo-hermaphroditism is presented. At no time was the real sex of this individual suspected. The condition was associated with a very marked cortical adrenal hyperplasia, with a very great increase of androgenic cells.

In the investigation of this case repeated careful attempts at perirenal insufflation of air proved valueless and misleading. The operative investigation of posterior exposure of both adrenal regions, advocated by Young, is undoubtedly the most satisfactory and a perfectly easy method of becoming aware of the whole pathological picture.

The operative findings showed a unilateral adrenal hyperplasia with absence of the other adrenal gland. The problem presented would appear to be almost without satisfactory surgical solution, and accidental death was probably a fortunate event.

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THE TECHNIQUE OF BRACHIAL PLEXUS BLOCK ANÆSTHESIA

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INTRODUCTION

BRACHIAL plexus block anæsthesia was originally devised by Kulenkampff in 1911, but in spite of many individual advocates it has never attained any degree of popularity. Most surgeons who have tried it have found the anæsthesia produced to be often unsatisfactory, and even the best statistics given do not exceed 60 per cent to 80 per cent of satisfactory results.[10, 15, 17] It is no wonder, therefore, that this type of anæsthesia should be regarded with such disfavour.

The methods of producing brachial plexus anæsthesia can be divided into four groups: (1) Supraclavicular route, devised by Kulenkampff; (2) Infraclavicular route, devised by Louis Bazy; (3) Axillary route, devised by Hirschel; (4) Paravertebral route, devised by Kappis.

Of these the supraclavicular route has been most widely employed, but even by this route the difficulty of obtaining uniformly perfect anæsthesia is shown by the large variety of techniques which have been described. Some insist that the plexus must be penetrated by the needle,[3] others condemn this as dangerous[8]; some take the subclavian artery as their guide to the plexus, while others rely on a standard surface-marking. Twenty to thirty cubic centimetres of 2 per cent novocain is the volume usually recommended for injection.

I have experimented with the various published methods on some fifty patients, but I have found the anæsthesia obtained to be often imperfect. Eventually I developed a technique which has only once failed to give perfect anæsthesia in my last 44 consecutive cases.

SURGICAL ANATOMY OF THE BRACHIAL PLEXUS

Where the brachial plexus crosses the first rib it consists of an upper, middle, and lower trunk. Anterior to the lower trunk lies the third part of the subclavian artery. The plexus and artery lie in a fascial investment represented by two walls: in front, a layer of fascia extending across from the scalenus anticus; and behind, a layer of fascia covering the scalenus medius.

It is said that for permeation of the nerve-trunks to occur the injection must be made between these two fascial layers, for the fascia is impermeable to the anæsthetic solution.[8] The subclavian vein lies in front of and at a lower level than the subclavian artery. The exact surface-marking of the brachial plexus appears to be somewhat variable, and Rhone has stated that post-mortem injection of dye with a needle introduced in the calculated direction may occasionally show that the needle point is as far away as 3 cm. from the plexus.[15] It must, however, be exceptional to miss the plexus by so wide a margin.

INDICATIONS AND CONTRA-INDICATIONS

Any operations on the upper extremity can be done under brachial plexus anæsthesia. In the series of 44 cases upon which this article is based, there were 2 cases of extensive operations on the shoulder-joint, one of them an amputation through the shoulder. In these high operations an infiltration round the supraclavicular nerves and the intercosto-humeral nerve has, of course, also to be made. Most of the operations done, however, were for various fractures in the region of the elbow.

I have avoided plexus anæsthesia in two types of case: (1) Where sepsis of the arm was present. It has been claimed that it is quite safe in such cases,[7] but I have a constant fear of a supraclavicular infection following the injection of a volume of fluid in such close proximity to the lymph-drainage area of the arm. (2) Where a more simple infiltration in the distal part of the arm would suffice (e.g., injection round the base of a finger).

It is frequently stressed that some degree of intelligent co-operation on the part of the patient is essential when injecting the plexus,[3] but, using the method described below, all that is demanded is that the patient should lie quiet while the injection is being given; the surgeon is not dependent on the subjective symptoms of the patient in deciding when the injection should be made. The penetration of the plexus by the needle is not very painful, for the pain threshold of nerve-trunks is much lower than that of the nerve-endings in the skin.

Age.—There is no maximum age limit: my youngest patient was 8 years old. If a child is given a small dose of morphia beforehand and the arm made absolutely devoid of sensation, there is no difficulty in keeping him quiet during the operation; in fact, I have found brachial plexus anæsthesia in children particularly easy to obtain.

TECHNIQUE

Premedication.—A hypodermic injection of $\frac{1}{4}$ gr. morphia is given half-an-hour before the patient is brought to the theatre; though nearly always wide awake on arrival, the patient frequently falls asleep once the plexus block has been given and the operation started.

Apparatus.—(*a*) One 10-c.c. Record syringe; (*b*) One hypodermic needle; (*c*) One needle of 20 gauge and $2\frac{3}{8}$ in. long; (*d*) 60–80 c.c. of 2 per cent planocain and 6 min. of 1–1000 adrenaline.

Position of Patient.—The patient lies supine on the operating table with the shoulder depressed and the head turned to the opposite side.

Making the Injection.—Placing the thumb and middle finger of the left hand on the acromioclavicular and sternoclavicular joints respectively, determine the midpoint of the upper border of the clavicle and raise a small intradermal wheal there, using the hypodermic needle and a little of the 2 per cent planocain. Then take the long needle and insert it through the wheal, aiming roughly in the direction of the second or third dorsal spine. The needle is inserted slowly without the syringe attached, and its direction altered as required until its point can be felt to impact on the first rib. The needle should now lie well above and lateral to the brachial plexus.

Should any blood come through the needle during its insertion, it must be withdrawn and a second insertion made. The syringe, loaded with 2 per cent planocain-adrenaline solution, is then attached and an injection of 5–10 c.c. made as the needle is withdrawn until its point lies in the subcutaneous tissues. I have found it convenient to hold the piston stationary and withdraw the barrel of the syringe with the needle; the 5–10 c.c. of anæsthetic solution is thus evenly distributed along the track of the needle. The syringe is now detached and the needle re-inserted so as to hit the first rib 5 mm. nearer its sternal end. Not until contact is made between the needle point and the first rib (no blood having been observed to issue from the needle as it was slowly inserted) is the syringe attached

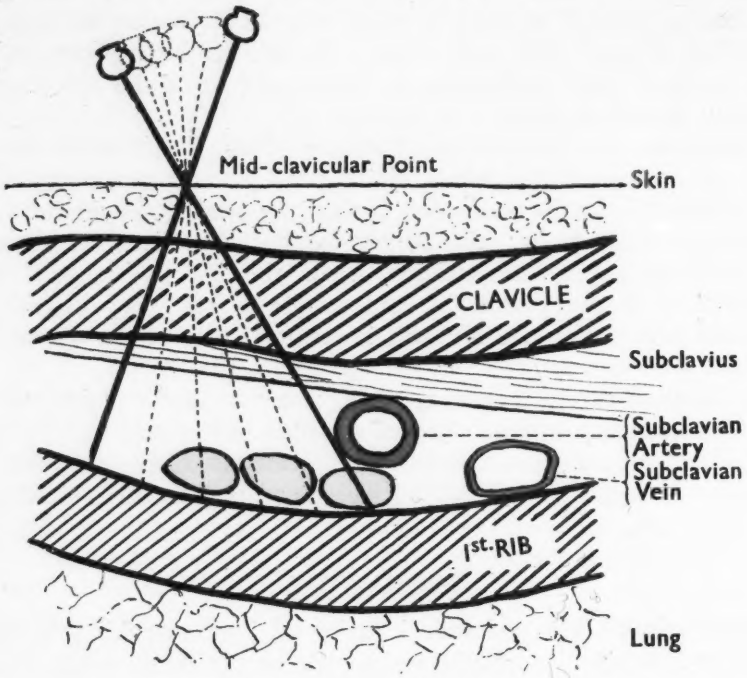

FIG. 545.—Diagram to show the relations of the brachial plexus and the directions in which the injections are made; the three trunks of the plexus are coloured yellow.

and a further 5–10 c.c. of anæsthetic solution injected. The injections are thus repeated, altering the needle axis each time so that the needle point progresses 5 mm. farther towards the sternal end of the rib. By thus systematically working one's way down, the sector of tissue between the midclavicular point and the first rib, across which the brachial plexus passes, is thoroughly infiltrated. The injections start outside the plexus, but how is one to determine when the lower trunk of the plexus which lies behind the subclavian artery is reached? The adequate infiltration of this trunk is the part of the procedure which is so often imperfectly done.

If the needle puncture in the skin is made at the mid-clavicular point, it will be found that, as the injections proceed, the obliquity of the needle has to be gradually altered in order that the needle point may impinge on the first rib each time; thus a point is finally reached at which the needle penetrates or lies close to the lower trunk of the brachial plexus (*Fig.* 545). The shaft of the needle at

this stage lies against the upper and outer aspects of the third part of the subclavian artery and the pulsation of the artery is transmitted to the needle, so that the hilt can be seen beating synchronously with the artery. When this is observed, the final injection is made. In all, five or six injections are required before the artery is reached.

Occasionally the first evidence of the proximity of the needle to the subclavian artery is the welling up of arterial blood along the lumen of the needle; using a needle of the bore recommended, the blood is never found to spurt when the artery is punctured. Puncture of the artery would appear to be absolutely harmless; I have done it in 8 of my 44 cases, and in a good many more of my earlier cases, but I have never seen any trouble ensue. To inject the lower trunk with certainty one must not be afraid of puncturing the artery. If the syringe is not attached, and it never should be while inserting the needle, blood appears immediately should the subclavian artery be accidentally penetrated; it is then an easy matter to withdraw and re-insert the needle so that its point passes just outside the artery and comes to rest against the first rib, posterior to the artery, and therefore in the lower trunk of the brachial plexus.

During the injections the three nerve-trunks are nearly always punctured in succession. The patient feels some tingling down the arm, but it is remarkable how seldom a muscle twitch is produced. The angle which the clavicle makes with the first rib is variable and one occasionally experiences some difficulty in striking the first rib with the needle prior to each injection, particularly in the more anterior part of the rib where it lies well under cover of the clavicle. The total volume of 2 per cent planocain injected is on an average 60 to 70 c.c. This is two to three times the volume of solution recommended by other authors.

RESULTS

The series of consecutive cases done by this technique numbers 44. Of these 43 were completely successful—that is to say, there was never a murmur of complaint from the patient during the operation, and on being questioned afterwards he stated that he had felt no pain whatsoever. In the 1 case which I considered a failure (an operation on a fractured humerus), the operation had been half completed before the patient began to complain of pain, and a general anæsthetic was administered.

Degree and Duration of Anaesthesia.—Complete anæsthesia is sometimes present immediately after the injection has been made, but usually it takes up to ten minutes to develop fully. Pain sensation is the first to go; very occasionally some degree of touch and pressure sense remains; motor power is always weakened, but in only 5 of my cases was it completely gone. In the majority of cases the fingers could still be moved voluntarily, but there was not enough muscular power left to lift the arm or hand against gravity. The anæsthesia remains complete for from 1½ to 2 hours, and then passes off slowly.

DANGERS AND COMPLICATIONS

The only complication which I have had was a massive collapse of the lower lobe of the right lung coming on two days after an operation for a compound fracture into the elbow-joint. The collapse occurred on the side opposite to the

plexus block and therefore probably had no relation to the type of anæsthesia used. The following complications have, however, been described :—

1. During Injection.—

a. Injection of Anæsthetic Solution into a Vein.—The injection of even a few cubic centimetres of the solution into a vein at the base of the neck may be fatal.[7] This danger can be avoided by always introducing the needle slowly, without the syringe attached; if blood appears while inserting the needle, the latter must be withdrawn and re-inserted in a slightly different direction.

b. Puncture of the Subclavian Artery.—Using a fine needle, puncture of the artery is harmless.

c. Puncture of the Pleura or Lung.—This must frequently happen while probing for the first rib, but I have never seen any harm result. Cases of pleural shock and of pneumothorax have, however, been described.

d. Temporary Paralysis of the Phrenic Nerve.—This is probably a common occurrence, especially when injecting the relatively large volume of solution which I employ. It apparently gives rise to no symptoms.

e. Paralysis of the Cervical Sympathetic.—This is said to occur sometimes, but I have never observed any evidence of it.

2. Later Complications.—

a. Toxic Symptoms from Absorption of Local Anæsthetic.[7]—I have observed no toxic symptoms following injection of up to 80 c.c. of 2 per cent planocain in adults. The addition of 6 minims of adrenaline to the anæsthetic solution will certainly delay its absorption as well as prolong the analgesia.

b. Massive Collapse of the Lung.—This may be a sequel to temporary phrenic nerve paralysis, but, as mentioned above, the only case in which I encountered this complication developed the collapse on the side opposite to the plexus anæsthesia.

c. Permanent Paralysis.—Paralysis of any degree has never been observed or even suspected in any of my cases. A few cases have, however, been reported,[10, 7, 14] but it is almost certain that these were due to damage inflicted on the plexus by indiscriminate prodding with too large a needle.[5] Cases of brachial plexus neuritis have also been reported, but this also is probably attributable to undue trauma with the needle.

In conclusion, I should like to add that the technique described is easily acquired, and in fact a good number of brachial plexus blocks have thus been successfully done by various house-surgeons and casualty officers in the Sheffield Royal Hospital.

SUMMARY

1. A technique of supraclavicular brachial plexus block anæsthesia is described and a consecutive series of 44 cases reported, with only 1 failure.

2. The degree of anæsthesia and its duration are described.

3. Reference is made to the reported dangers and complications of brachial plexus anæsthesia.

I have to thank Mr. H. Blacow Yates, Honorary Surgeon to the Sheffield Royal Hospital, for his permission to carry out this work on his cases, and for his helpful criticism of this paper. My thanks are also due to Mr. J. B. Ferguson Wilson, Mr. J. C. Anderson, and Mr. Wilfred Hynes, Honorary Surgeons to the

Sheffield Royal Hospital, on some of whose cases my first attempts at brachial plexus anæsthesia were done. One of the cases in the series was a demonstration case done at the request of Mr. H. A. Brittain, Orthopædic Surgeon to the Norfolk and Norwich Hospital, and I have to thank him for the privilege.

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CANCER OF THE STOMACH AS A SURGICAL PROBLEM *

By REGINALD T. PAYNE, London

Cancer of the stomach is a disease of appalling frequency in the community. Its victims come under observation at a relatively late stage, and at the present time surgery alone offers the possibility of cure. Only a very small proportion of the total cases is subjected to radical surgical measures, and of the cases which survive operation very few are alive at the end of the five-year period. As a biological problem, the frequency of cancer of the stomach is important, since it ranks high among killing diseases. At the same time, the fact that operative treatment alone offers a possibility of cure renders the problem of treatment an ever present one to the surgeon. If improvements in the treatment of this disease are to be achieved, they will only come as the result of concentration on, and constant review of, the problems inherent in the subject.

During recent years many important contributions have been made to the knowledge of diseases of the stomach. These advances have embraced physiology, pathology, radiology, biochemistry, hæmatology, and the ancillary methods of investigation. They are being only slowly incorporated in current surgical practice, and it is still true to say that gastric surgery in general, including cancer surgery, often concentrates more on the morbid anatomical features of disease than on the more general aspects of the pathology of the stomach.

This paper is a survey of the problem of cancer of the stomach mainly from the point of view of the surgeon. This method of approach requires a consideration of the incidence of the disease in the community; a survey of a large amount of clinical material; a consideration of some of the problems of pathology and physiology; a consideration of some of the clinical problems; and a survey of some of the problems of operative treatment itself. Operative technique, however, has not been dealt with in detail. A survey of this kind necessitates the introduction of a good deal of statistical material. Whilst fully realizing the limits of usefulness of the statistical method, the fallacies which are bound to occur, and the difficulties arising from lack of uniformity of approach, yet I am sure that a statistical approach is essential to an appreciation of the gravity of the disease and of its problems. In surgery there is no short cut to success, and in dealing with a serious problem such as cancer of the stomach it is only by constantly reviewing the sources of information, and by incorporating newer knowledge as it becomes available, that improvement in results is likely to be obtained.

THE INCIDENCE AND IMPORTANCE OF THE DISEASE

Incidence.—The importance of cancer of the stomach lies in its frequency. During the year 1933 there were 11,929 deaths from the disease in England and

* A Hunterian Lecture given at the Royal College of Surgeons of England, on January 19, 1938. Based on work done in the Departments of Pathology and Surgery at St. Bartholomew's Hospital, and the Department of Surgery at the British Post-Graduate Medical School.

Wales.[1] In the United States it is estimated that approximately 38,000 persons die annually from this cause (Eusterman and Balfour[2]). In this form of cancer, as in the greater numbers of other forms, the statistics of most countries show a considerable rise in the incidence during recent years. It is difficult to determine whether or not there has been any real increase in incidence, since it is certain that the greater accuracy in diagnosis and in the compilation of mortality tables, and the increase in the population living beyond the age of 40, have all been contributory factors in the apparent increase.

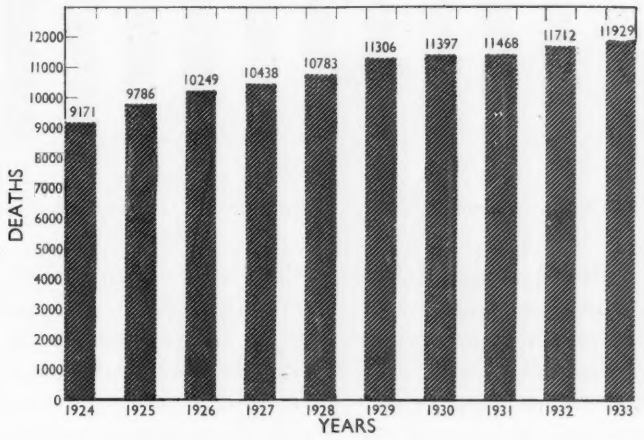

FIG. 546.—Carcinoma of stomach. Annual deaths of males and females, England and Wales, 1924–33.

It will be seen from *Fig.* 546 that during the decade 1924–33 there was an increase in the deaths registered in England and Wales from cancer of the stomach of approximately 30 per cent. This increase was shared equally by the sexes.

Sex.—The incidence of the disease is approximately the same in the two sexes, with a slightly greater incidence in males (*Figs.* 547, 548).

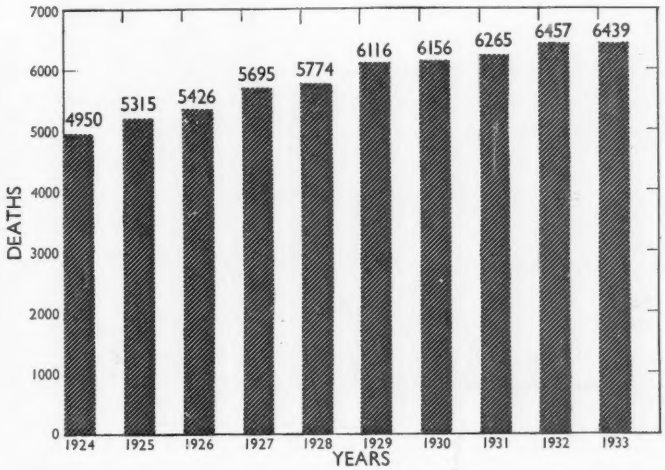

FIG. 547.—Carcinoma of stomach. Annual deaths of males, England and Wales, 1924–33.

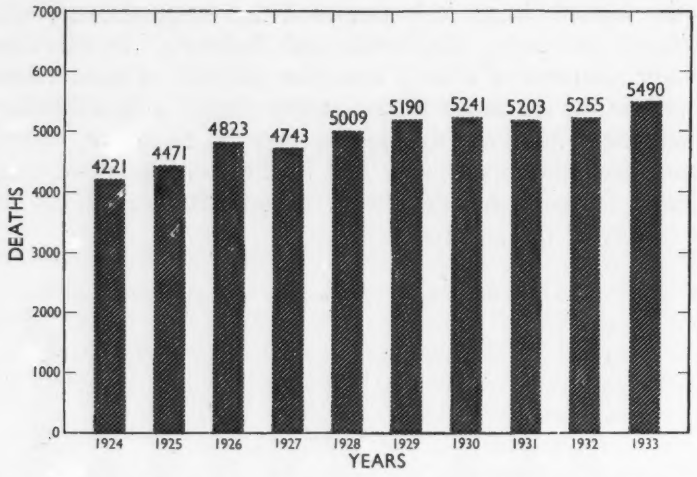

FIG. 548.—Carcinoma of stomach. Annual deaths of females, England and Wales, 1924–33.

Age.—The disease is not commonly met with under the age of 40, and it reaches its maximum incidence in the community between the ages of 65 and 70. When these figures are considered in relation to the numbers of the population living at the various age groups, it is obvious that cancer of the stomach becomes increasingly frequent with age. *Fig.* 549 shows the average annual death-rate from cancer of the stomach in England and Wales for the years 1924–33.

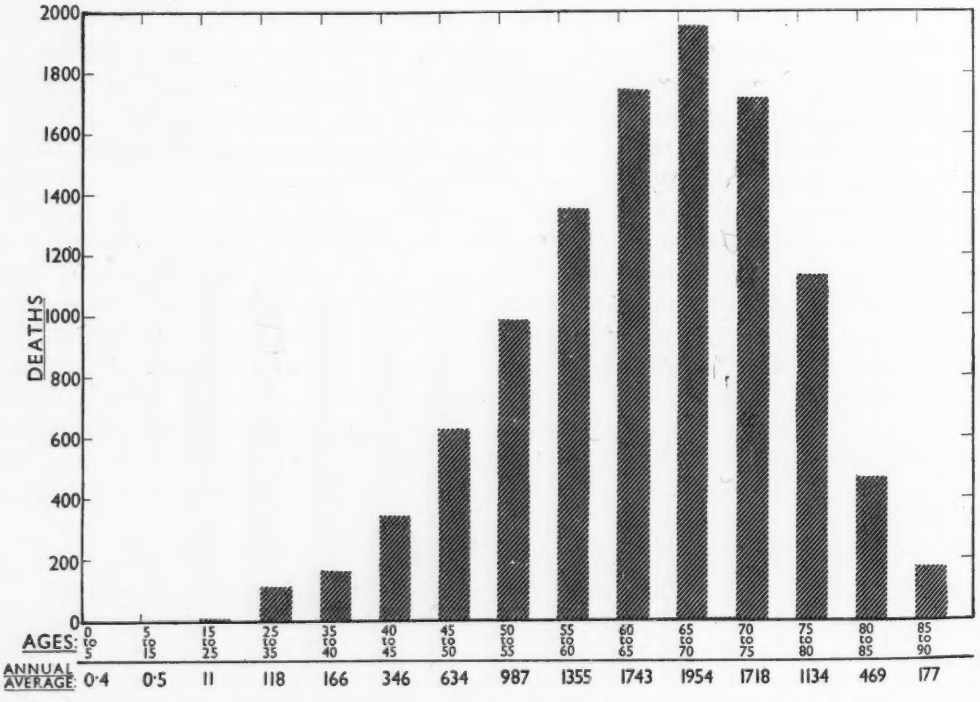

FIG. 549.—Carcinoma of stomach. Average annual death-rate, males and females, England and Wales, 1924–33.

Gastro-intestinal Cancer.—Cancer of the stomach accounts for approximately 42 per cent of cancers of the intestinal tract. During the year 1933 there were 28,064 deaths from cancer of the alimentary tract, and 11,929 of these, or 42·5 per cent, were of the stomach. Of every 100 cases of gastro-intestinal cancer, 4 were in the tongue, 8 in the œsophagus, 43 in the stomach, 27 in the large intestine, and 18 in the rectum. The incidence of cancer of the stomach is thus approximately equal to the combined incidence of cancer of the large intestine and of the rectum. (*Figs.* 550–552.)

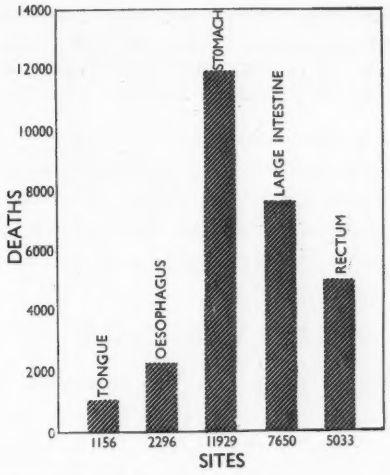

FIG. 550.—Sites and forms of fatal cancer of alimentary tract, males and females, England and Wales, 1933.

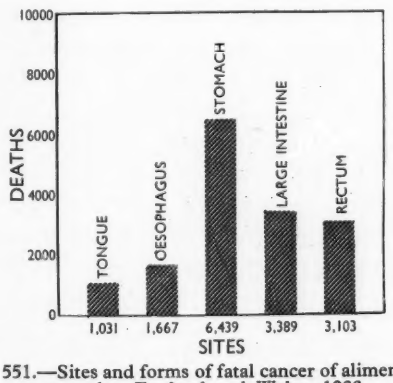

FIG. 551.—Sites and forms of fatal cancer of alimentary tract, males, England and Wales, 1933.

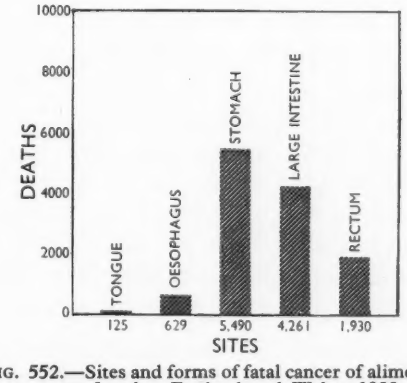

FIG. 552.—Sites and forms of fatal cancer of alimentary tract, females, England and Wales, 1933.

Table I.—SITES AND FORMS OF FATAL CANCER OF ALIMENTARY TRACT, 1933

Sites	*Males*	*Females*	*Total*
Tongue	1031 (6·6 per cent)	125 (1·0 per cent)	1156 (4·1 per cent)
Œsophagus	1667 (10·7 per cent)	629 (5·1 per cent)	2296 (8·2 per cent)
Stomach	6439 (41·2 per cent)	5490 (44·1 per cent)	11.929 (42·5 per cent)
Large intestine	3389 (21·7 per cent)	4261 (34·3 per cent)	7650 (27·3 per cent)
Rectum	3103 (19·8 per cent)	1930 (15·5 per cent)	5033 (17·9 per cent)
	15,629	12,435	28,064

From the figures in *Table I* it will be seen that the incidence of cancer of the stomach is approximately the same in the two sexes. Of the other major sites of gastro-intestinal cancer, the tongue, the œsophagus, and the rectum are all involved less frequently in women than in men. On the other hand, cancer of the large intestine is much commoner in women than in men.

Hurst, in his recent Harveian Oration,[3] states :—

> The total incidence of cancer is approximately the same in both sexes, in the rich and the poor, and in all nations. If cancer of the uterus and breast are excluded, the relative incidence in different organs is the same in men and women. It is also the same in the rich and the poor and in different nations, with one supremely important exception—the stomach being involved three times as often in the poor as in the well-to-do, and in only 22 per cent of all cases of cancer in men in Great Britain compared with 42 per cent in America, 55–60 per cent in Holland, Bavaria, Norway, Sweden, and Spain, and 66 per cent in Czechoslovakia.

If the figures from this country are considered for a moment, the vastness of the clinical problem becomes apparent. The suffering and misery produced by cancer of the stomach is colossal, and the public hospitals of the country have to shelter many of its victims in the terminal stages of the disease.

SURGERY THE ONLY CURE

With a disease of such frequency, the question of treatment becomes an urgent one. At the present time surgery in the form of either partial or complete gastrectomy offers the only possibility of cure. Other methods of treatment include irradiation by X rays, radium, and radon, and also the use of lead and other chemicals. These methods, however, can only be regarded as experimental or palliative, and they have not been responsible for any cures. When the surgery of the stomach is compared with the treatment of malignant disease in many other sites, it maintains its position unchallenged, since it has no alternative. The problem of the cure of cancer of the stomach is still the problem of the efficient surgical management and treatment of the disease. It is, however, a much greater problem than that of mere operative treatment, and an approach to it involves a consideration of all the factors which contribute to the failure of surgery to achieve its desired end. Some of the problems involved are those of gastric surgery as a whole rather than of cancer surgery in particular. Surgery may offer either palliation or cure, but as this paper is especially concerned with the question of the possibility of cure, I shall concentrate chiefly upon this aspect of the subject.

CLINICAL MATERIAL

For the purposes of this paper, all the cases of cancer of the stomach which were admitted to the wards of St. Bartholomew's Hospital during the years 1921–32 inclusive have been analysed :—

Cases analysed	506
Notes missing	48
Cases discarded on grounds of insufficient data or incorrect diagnosis	99
TOTAL	653

This series includes all cases both medical and surgical. Of the missing notes, 9 were gastrectomy cases, but there was nothing in this group as a whole to suggest that they were other than evenly distributed. Ninety-nine cases had to be discarded. In a few of these the diagnosis was obviously incorrect, but the majority were cases which had been admitted to the medical wards and had to be rejected on grounds of insufficiency of data. It is obvious, of course, that in discarding such notes a personal factor is involved, but at least this has been uniform. Many patients are admitted to hospital with vague abdominal symptoms which *may* be due to cancer of the stomach, but which lack the confirmation of detailed investigation, operation, or post-mortem examination. Such cases inevitably occur, and though for indexing purposes they may have to be classified under the heading of cancer of the stomach, it would not be justifiable to incorporate them here.

The figures which are quoted represent the work of all the physicians and all the surgeons during the period of this survey. No one individual had especially concentrated on gastric diseases in general, on gastric surgery, or on gastric cancer. The figures, therefore, represent the work of general physicians and general surgeons. For this reason, I believe they represent a much truer index of what is being done for cancer of the stomach in the community as a whole than do the figures provided by the work of one surgeon who has especially concentrated on gastric surgery or those from a clinic where the work is largely surgical. Whatever the advantages of the specialized method of approach to a problem, statistics derived from such sources are not likely to provide the most accurate basis for the provision of data relating to therapeutic measures in the community as a whole. The figures which are given do at least indicate the extent and nature of the problem of cancer of the stomach as it presents itself to a large teaching hospital which has avoided specialization, and they also give some indication of the clinical problem in the community.

In addition to the clinical data, material from 142 post-mortems upon patients dying with cancer of the stomach at St. Bartholomew's Hospital during the years 1919–36 has been investigated. For ten of these years, I was myself carrying out post-mortem work, and many of the cases actually passed through my hands.

THE PROBLEMS OF PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY

The debatable question of the aetiology of malignant disease of the stomach and the general pathological features of the disease will not be discussed, but certain aspects of the subject must be considered. Surgery in general has naturally always been largely influenced by the older morbid anatomists. Their claim, however, to represent the whole of pathology can no longer be maintained, and pathology to-day is vast when compared with merely descriptive morbid anatomy. In the case of malignant disease of the breast, adequate and successful surgery can be largely based on the morbid anatomy of the disease. In the case of malignant disease of the stomach the surgical approach must be on the widest pathological lines, and the surgeon who approaches the subject on purely morbid anatomical lines is certain to meet with failure. Cancer of the breast is, in the absence of metastases, rarely associated with any profound disturbances of nutrition

or metabolism. Cancer of the stomach may be associated with the most profound nutritional, metabolic, and hæmatological changes, even although the malignant process is still strictly confined to the stomach. Unless the functions of the stomach as a whole and their modification by malignant disease are adequately considered, there can be but little improvement in the surgery of gastric disease in general or of cancer in particular.

The following aspects of the pathology and pathological physiology of the stomach must be considered :—

1. Morbid Anatomy.—The chief morbid anatomical types of cancer of the stomach are well known and will not be considered in detail, but it is obvious that the site, size, and type of growth have important bearings upon the questions of gastric function and of operability. In certain situations, particularly the cardia and the pylorus, mechanical factors may be of outstanding importance in leading to gastric obstruction. In certain situations, as for example the cardia and fundus, the anatomical site may seriously hinder adequate surgical approach. These aspects of gross pathology are outside the scope of this paper.

2. Metastases.—The presence of other than merely local glandular metastases in relation to the stomach must of course inevitably bar successful surgery, though not necessarily contra-indicate palliative surgical procedures, including partial gastrectomy. The determination of the presence of such metastases is a clinical and an operative problem.

3. General Nutrition.—There is frequently profound impairment of the general nutrition of patients with cancer of the stomach. This is partly due to the loss of appetite and the impaired ingestion of food, partly to the loss of blood and anæmia, partly to the gastric obstruction, and partly to the toxæmia which may also be present. It is important to distinguish between general nutrition and mere loss of weight, and although these two are usually closely linked, they do not always run parallel. A small pyloric growth, for example, may lead to quite acute symptoms of obstruction and toxæmia due to perverted metabolism, without leading to any marked alteration in weight.

4. Weight and Weight Loss.—Rapid loss of weight is an important feature of cancer of the stomach, and losses of 2 st. in the course of six months are extremely common. In some cases the weight loss may be much more marked, but in the typical case it is rapid. In all cases it has important bearings upon the question of surgery. Studley[4] has recently analysed the bearings of antecedent weight loss upon the question of the surgical treatment of peptic ulceration. In *Table II* are sent out Studley's figures of percentage weight loss in comparison with the previously known best weight and in terms of operative mortality :—

Table II.—Peptic Ulceration : Pre-operative Percentage of Weight Loss and Post-operative Mortality. (*After Studley.*)

		Mean Percentage Weight Loss	*Number of Patients*	*Deaths*
Group A.	Those losing less than 20 per cent	12·6	28	1
Group B.	Those losing more than 20 per cent	26·1	18	6

It is obvious that in the case of simple peptic ulceration loss of weight has important bearings upon the question of successful surgery. I know of no comparable data concerning antecedent weight loss in connexion with the surgery and primary mortality of patients with cancer of the stomach, but I believe that the

relationship is a close one and is determined by the metabolic disturbances which accompany rapid loss of weight.

5. Amount and Nature of Food.—The nutritional disturbances in cancer of the stomach are largely determined by the inability of the patient to ingest an adequate amount of food. This deficient intake of food may be brought about by the actual loss of appetite, by the mechanical conditions present in the stomach, or by the disturbances of gastric function. Perversion of appetite may at times lead to a marked distaste for certain types of food. This is likely to be particularly marked in the case of proteins. But however these deficiences are brought about, the result is that the patient has often been living for many months on a diet hopelessly inadequate in quantity and quality for the normal metabolism of the body.

6. Vitamin Deficiency.—There is no doubt that many patients who have to take restricted dietaries on account of gastric disease often do not obtain adequate amounts of the various vitamins. It is difficult to be certain of the exact significance of these minor vitamin deficiencies which stop short of actual deficiency diseases, but the evidence strongly supports the view that such deficiencies diminish the resistance of the individual to infections. In a recent discussion at the Royal Society of Medicine on " Nutrition and its Effects on Infectious Disease ", Dr. Stuart J. Cowell[5] summarized the position as follows :—

> The general impression which one obtains from the kind of evidence outlined here—and I hope that the selection of observations may be regarded as a fair one—is that it is exceedingly difficult to prove definitely the relation of any particular food factor to resistance to infections in man. One is left with the feeling that in so far as the state of nutrition is connected with resistance to infection, the connexion is rather that good nutrition may determine the course an infection will take than that it will decide whether infection will occur. The evidence available at present in the case of man does not seem to warrant the view that one isolated food constituent is responsible in any specific way for maintaining the resistance of the body to infection in any general sense. On the other hand, there is suggestive evidence that gross malnutrition in a broad sense of the term, or individual states of specific food deficiency, may lower the resistance to established infections. The possibility will have to be considered that an optimum state of nutrition, lasting for a longer time in the life of the individual than has been aimed at in most of the tests so far reported, may be necessary to secure the maximum degree of resistance to infections.

In the same discussion Dr. Harriett Chick[6] quoted the summary of Clausen: " Susceptibility to infection is not as a rule affected by diet; resistance to infection, on the other hand, may be greatly reduced by deficient diet."

Certainly the clinical evidence suggests that patients with gastric disease are often obtaining deficient amounts of vitamin C, and the experimental evidence suggests that this vitamin is essential to the production of scar tissue. Peripheral neuritis may occasionally develop in cases with pyloric obstruction due to a deficiency of vitamin B_1 (Laurent and Sinclair[7]).

It is impossible to separate the vitamin factors from the general nutritional factors in cancer of the stomach, but it is probable that the greatly lowered resistance to infection of these patients is determined partly at least by the deficient vitamin intake.

7. Fluid Balance and Dehydration.—In cancer of the stomach there is frequently a deficient water intake, or actual loss of fluid through vomiting. The patient thus suffers from some degree or other of dehydration which is likely to be most marked in association with severe vomiting. The urinary output is often

greatly diminished in an effort to conserve body fluids. Some patients only pass urine two or three times during the course of twenty-four hours. In the absence of vomiting an adult lying in bed requires approximately 1500 c.c. of fluid a day for adequate urine excretion, and an equal amount for the insensible loss of water in the sweat, respiration, and stool. The majority of patients with cancer of the stomach certainly get nothing like this amount of fluid during the course of twenty-four hours.

8. The Effect of Gastric Obstruction and Vomiting.—Falconer and Lyall,[8] in their recent article on "Blood Chemistry in Intestinal Obstruction", summarize the chemical effects of high intestinal obstruction as follows:—

> It is now generally accepted (Haden and Orr, 1923, 1926; Orr, 1936) that during experimental high intestinal obstruction in animals there occurs a fall in the blood-chloride values, a rise in the blood-urea and non-protein-nitrogen values, an increase in the CO_2-combining power of the plasma, and an increase in the concentration of the blood. Clinical cases of high intestinal obstruction of such types as pyloric stenosis with vomiting (Brown et al., 1923), hypertrophic pyloric stenosis of infants (Graham and Morris, 1929), vomiting after gastro-enterostomy (Haden and Orr, 1923*b*), and upper jejunal obstruction (Haden and Orr, 1923*c*), have all been found to show hypochloræmia, azotæmia, alkalosis, and dehydration, while Cooke (1932) reported similar changes in the alkalosis that occurs during the treatment of peptic ulcer by alkalis in susceptible subjects.

These specific chemical factors are considered briefly in the succeeding sections.

9. Hypochloræmia.—Hypochloræmia is caused by the loss of chlorine ions as the result of vomiting, and in general the fall in the plasma chloride is determined by the severity and duration of the vomiting.

10. Plasma CO_2-combining Power.—In association with the fall in the blood-chlorides there is a disturbance of the acid–base equilibrium, with a tendency towards alkalosis.

11. Azotæmia.—Falconer and Lyall[8] point out that a rise in the blood-urea is the only biochemical change that is constantly found in obstruction at all levels. The evidence in this connexion indicates that in obstructive conditions there is an increase in endogenous protein metabolism. In the case of cancer of the stomach, the partial starvation and the gastric obstruction are both factors tending to produce azotæmia.

12. Achlorhydria.—The deficiency or even absence of hydrochloric acid in the gastric secretion has important bearings upon many of the pathological features of the disease. From the standpoint of surgery, however, the loss of the antiseptic barrier of the stomach is the most serious. In the absence of acid secretion there is a marked increase in the level of the intestinal flora, and colonic organisms may reach the stomach. The restricted dietary and the relative absence of chewing lead to a great increase in the buccal flora, and these swallowed organisms are no longer subjected to the bactericidal action of the gastric juice. Finally, the presence of a growth in the stomach and the associated stasis render the whole of the upper alimentary tract a much more dangerous field of operation than is the case in health.

13. Anæmia.—Anæmia is a characteristic feature of practically all cases of cancer of the stomach. In general, it is of the microcytic variety and results from the poor dietary, from the achlorhydria with its resulting impairment of iron absorption, and from the actual loss of blood from the surface of the growth. The type of anæmia does not seriously differ from the other varieties of microcytic anæmia.

Macrocytic anæmia occasionally occurs in association with cancer of the stomach, but it is exceptionally rare. Its importance has, perhaps, been overstressed since cases of this type are more likely to find their way into the literature. In such cases there is a deficiency of the intrinsic factor. Other nutritional factors may of course be present.

In cases in which there are secondary deposits in the bones, immature red and white cells may appear in the peripheral circulation. A negative blood-finding does not, however, exclude the presence of such deposits.

The stomach is so important an organ for the well-being of the whole body that a disturbance of one function may well affect the other functions. Cancer of the stomach must be regarded not only as a local malignant process with all the characteristics of malignant disease, but also as a disease process which, by interference with the fundamental and diverse functions of the stomach, may profoundly influence nutrition and metabolism as a whole. The combined problems of the surgery of cancer of the stomach can only be viewed in proper perspective if due regard is paid to the wider aspects of gastric function, and if morbid anatomical factors are not allowed completely to dominate the picture.

CLINICAL AND DIAGNOSTIC PROBLEMS

The various clinical manifestations of cancer of the stomach will not be discussed in this paper, but reference must be made to the protean and vague nature of many of the early symptoms. There is no one symptom or group of symptoms which is necessarily characteristic of cancer of the stomach, and this is an important factor in making the early recognition of the disease so difficult. Large areas of the stomach are relatively silent, particularly the fundus and the greater curve, and a disease which may start so vaguely or with such varying clinical syndromes may well remain unsuspected in the early stages. The diagnostic difficulty which is present in the primary cases is even greater in the cases which develop secondarily to gastric ulceration or follow antecedent dyspepsia. In ulcer-cancer, the symptoms of transition may be so insidious and so gradual that clinical recognition of the change is practically impossible.

Symptoms referable to the stomach are present at some time or other in the majority of patients with cancer of the stomach, and there are few organs in the body which the patient feels so well qualified to treat as his own stomach. In this he is encouraged by the advertisements of stomach remedies which abound, and by the relief which may come from these, even in malignant disease. In addition, the frequency of all types of digestive disorders and the variety of diseases which may be referred to the stomach tend to throw the medical adviser off his guard. There can be no really early diagnosis of cancer of the stomach until the exact diagnosis of ' indigestion ' is taken seriously both by the patient and by his doctor, and every case arising over 40 years of age subjected to detailed investigation. Surgery cannot achieve a great deal if in the average primary case many months elapse between the onset of symptoms and the patient's admission to hospital.

This exact diagnosis of cancer of the stomach can only be built up by careful clinical examination and on the data provided by the test-meal, occult blood

test, radiology, gastroscopy, hæmatology, etc. Of these, radiology is still the most generally applicable, the most certain, and the safest, but there are fallacies and errors. Finally, in the absence of gross metastasis, exploratory operation must be undertaken for diagnostic purposes.

ANALYSIS OF CLINICAL DATA

(*St. Bartholomew's Hospital*, 1921–32)

During the years 1921–32 inclusive, 506 cases of cancer of the stomach were admitted to the wards of St. Bartholomew's Hospital. These cases form the basis of the present analysis.

Sex Incidence.—Of the 506 cases in the present series, 330, or 65 per cent, were males and 176, or 35 per cent, were females.

Age Incidence.—The following were the age incidences of the cases :—

Table III.—Age Incidence of Cancer of Stomach

Ages	*Cases*
15–20	2
20–25	1
25–30	9
30–35	11
35–40	25
40–45	60
45–50	70
50–55	89
55–60	104
60–65	71
65–70	44
70–75	17
75–80	3

It will be seen that no five-year period over the age of 15 was entirely exempt, but that the majority of cases occurred during the decade 50–60 (193 cases) and that in the two decades 40–60, 323 cases occurred. The two youngest patients in the series were aged 17 and 18, and there were 23 cases under the age of 35, and 48 cases under the age of 40. The under 40 cases thus account for approximately 10 per cent of the total of 506 cases.

Length of History.—In a considerable number of cases there was an antecedent history of indigestion or peptic ulceration, and the symptoms attributable

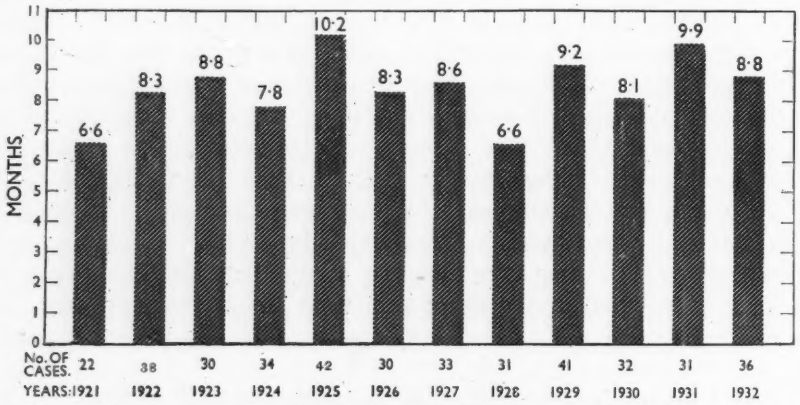

Fig. 553.—Carcinoma of stomach. Average duration of symptoms in 400 under-two-year cases, St. Bartholomew's Hospital, 1921–32.

to these gradually merged into those of cancer of the stomach. For the purposes of analysis the cases have been divided into those in which the length of history did not exceed two years and those in which it did. The under-two-year group may, I think, be justifiably considered to comprise all the cancers which arose without antecedent peptic ulceration. Of the total 506 cases, the history was under two years in 400 cases, or approximately 80 per cent. For this purpose the length of history has been taken to extend from the onset of symptoms to the time of the patient's admission to hospital. The over-two-year group has not been considered in detail.

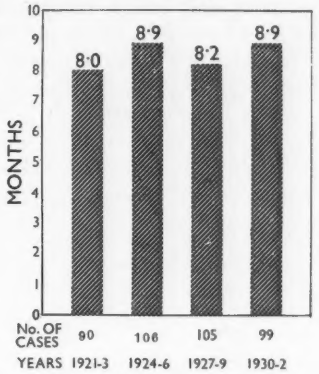

FIG. 554.—Carcinoma of the stomach. Average duration of symptoms in 400 under-two-year cases; in three-yearly periods, St. Bartholomew's Hospital, 1921–32.

If the cases in the under-two-year group are analysed in yearly periods, the shortest average symptom duration was 6·6 months and the longest average 10·2 months (*Fig.* 553). If the cases are further analysed into three-yearly periods, they display variations from 8·0 months (1921–3) to 8·9 months (1924–6 and 1930–2) (*Fig.* 554). There is thus nothing in these figures to suggest that during the 12-year period under review there was any shortening

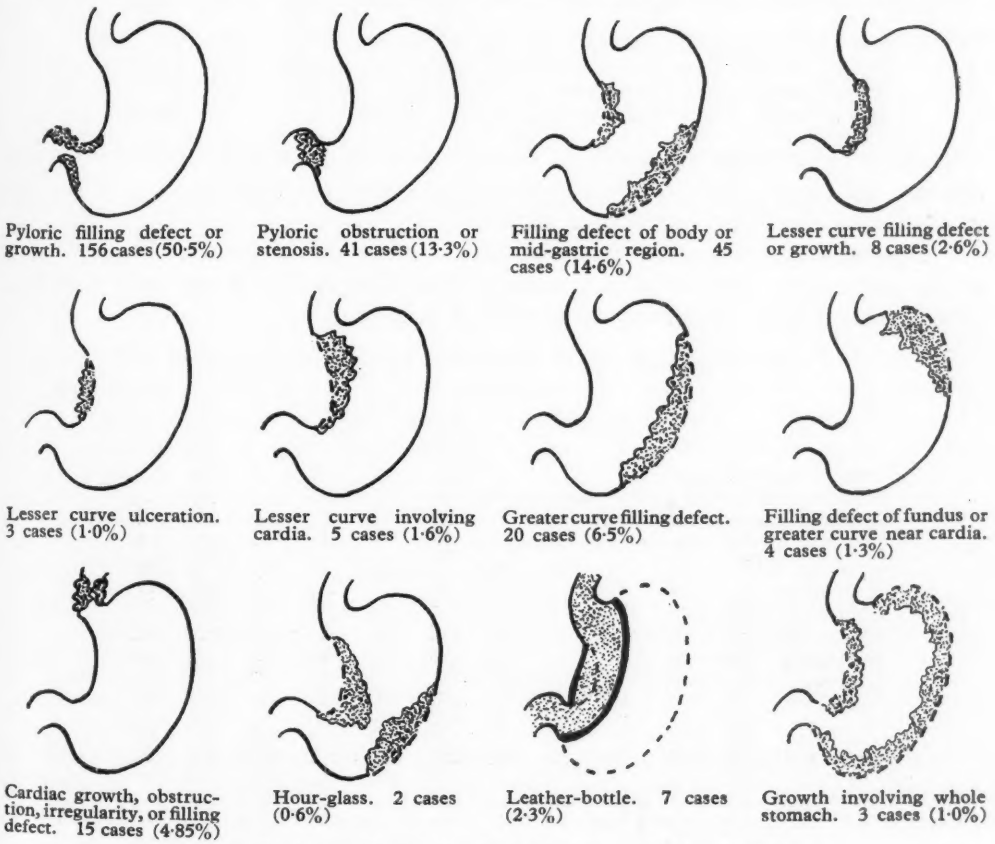
Pyloric filling defect or growth. 156 cases (50·5%)
Pyloric obstruction or stenosis. 41 cases (13·3%)
Filling defect of body or mid-gastric region. 45 cases (14·6%)
Lesser curve filling defect or growth. 8 cases (2·6%)
Lesser curve ulceration. 3 cases (1·0%)
Lesser curve involving cardia. 5 cases (1·6%)
Greater curve filling defect. 20 cases (6·5%)
Filling defect of fundus or greater curve near cardia. 4 cases (1·3%)
Cardiac growth, obstruction, irregularity, or filling defect. 15 cases (4·85%)
Hour-glass. 2 cases (0·6%)
Leather-bottle. 7 cases (2·3%)
Growth involving whole stomach. 3 cases (1·0%)

FIG. 555.—Carcinoma of stomach. Analysis of X-ray findings in 309 cases.

of the period before the patient with cancer of the stomach came under observation.

In the series of 400 under-two-year cases, there were only 67 in which the symptoms were under three months.

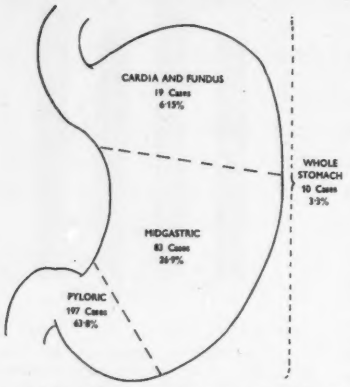

FIG. 556.—Carcinoma of stomach. Summary of X-ray findings in 309 cases.

The under-two-year group is thus numerically much the larger, and accounts for approximately 80 per cent of the total number of cases. It is important to recognize that in this large group there had been no antecedent history of any type of dyspepsia whatever, and that the symptoms due to the cancer of the stomach were the first dyspeptic symptoms from which the patient had suffered. The majority of cases of cancer of the stomach thus arise in patients in whom the upper digestive tract has never given any serious cause for anxiety.

Radiology.—Radiological investigations provided a clue to the frequency of growths at the various sites in the stomach. Detailed analyses were available in 309 of the cases, and are set out in the accompanying diagrams (*Figs.* 555, 556).

The following table summarizes the distribution :—

In	19 cases, the growth involved the cardia and fundus	6·15	per cent
In	83 cases, the growth involved the midgastric region	26·9	per cent
In	197 cases, the growth involved the pyloric region	63·8	per cent
In	10 cases, the growth involved the whole stomach	3·3	per cent

Thus in approximately two-thirds of the 309 cases the growth involved the pyloric region. Of the 20 cases involving the greater curve, all were filling defects, and in none was there excavation or a niche.

Operability.—Operative treatment was carried out in 351, or 68·4 per cent, of the 506 cases. This includes 3 cases in which the operation was only a biopsy. The details of these operations are set out as follows :—

Table IV.—ANALYSIS OF OPERATIVE TREATMENT IN CANCER OF STOMACH

Operation	*No. of Cases*	*No. of Deaths*	*Mortality*
Partial gastrectomy	54	18	33·3 per cent
Local excision and gastro-enterostomy	1	—	—
Anterior gastro-enterostomy	45	23	51·1 per cent
Posterior gastro-enterostomy	93	30	32·25 per cent
Gastrostomy	5	1	20·0 per cent
Jejunostomy	6	4	66·6 per cent
Entero-enterostomy	1	—	—
Closure of perforation	6	6	100·0 per cent
Exploratory laparatomy	136	29	21·3 per cent
Exploratory laparotomy and drainage	1	1	100·0 per cent
Biopsy	3	1	33·3 per cent

It will be seen that radical operative treatment in the form of partial gastrectomy was carried out in 54 of these 506 cases, giving a percentage of 10·7. In approximately only one in ten, therefore, of the cases of cancer of the stomach presenting themselves at the hospital was any radical treatment possible.

The nature of these operations is set out graphically in *Fig.* 559.

Mortality.—Of the 351 cases which were subjected to operation, the result was fatal in 113, or 32·5 per cent. The total mortality is set out in *Fig.* 558, and is discussed further in the section dealing with post-operative mortality.

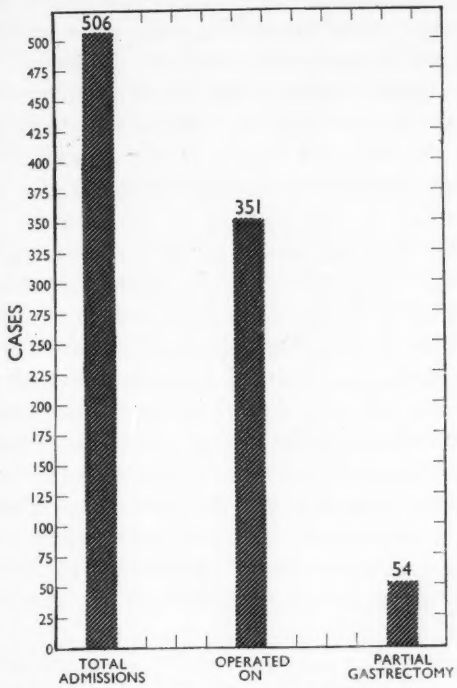

FIG. 557.—Carcinoma of stomach. Admissions, operations, and partial gastrectomies, St. Bartholomew's Hospital, 1921–32.

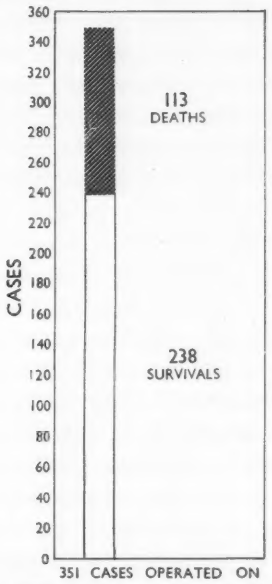

FIG. 558.—Carcinoma of stomach. Mortality of cases operated on, St. Bartholomew's Hospital, 1921–32.

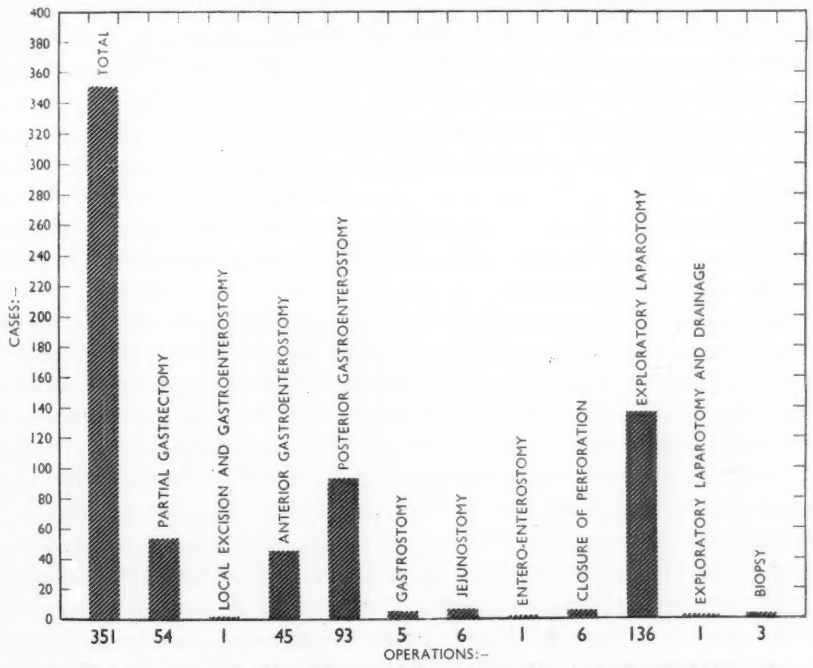

FIG. 559.—Carcinoma of stomach. Analysis of 351 operations, St. Bartholomew's Hospital, 1921–32.

THE SURGICAL PROBLEMS

What are the main problems which confront the surgeon when a diagnosis or probable diagnosis of cancer of the stomach is made? These problems may be considered under the headings of pre-operative, operative, and post-operative, and also in relation to radical surgery and to palliative surgery. Some of the factors involved are controllable; others again, such as the morbid anatomical ones, are completely beyond the control of the surgeon. With the technical limitations imposed upon the surgeon by the site and nature of the disease, how far can the controllable factors be used to improve results and how far can the post-operative mortality be reduced?

Operative intervention in cancer of the stomach is always a major operative procedure and it carries with it many risks. These risks are the risks of operative intervention in a patient with a rapidly developing and profound nutritional, metabolic, and blood disturbance. Apart from actual technical errors, the immediate risks are chiefly bacterial ones due to the increased bacterial activity in the upper alimentary tract and the lowered resistance to infection due to the gross nutritional impairment. The majority of deaths after radical, palliative, or exploratory operations in patients with cancer of the stomach arise from infection either of the peritoneum or of the lungs. All the evidence suggests that the frequency of such infections resides in the patient's inability to put up any real resistance to any type of infection however mild. In a series of 51 cases of partial gastrectomy for either ulcer or carcinoma which came to post-mortem, infection accounted for approximately 70 per cent of the deaths (Payne[9]).

Saltzstein and Sandweiss,[10] in reviewing similar data for cancer of the stomach, found 60 per cent of the deaths were due to peritonitis and pulmonary infections. It is probably true that many of the deaths early in the post-operative period are indirectly as much attributable to the antecedent toxæmia, nutritional disturbance, dehydration, or alkalosis, as to the trauma of the operation and of the anæsthetic. Infection is the determining factor leading to death, but it operates against a background of gross nutritional impairment.

The importance of this background of nutritional disturbance is supported by the high mortality from infection following exploratory laparotomy or gastro-enterostomy in patients with cancer of the stomach. But of course the cases in which radical surgery cannot be carried out represent the more advanced types of the disease and therefore the poorer operative risks. Statistics bearing upon this have already been quoted.

Pre-operative Preparation.—Except in the case of actual perforation of a growth, operations for cancer of the stomach should never be carried out as emergencies. And it is true to say that the presence of acute or subacute gastric obstruction should be a definite contra-indication to early operative treatment. Time spent on pre-operative treatment of these cases is time well spent. A delay of ten to fourteen days may even be justified; for the advantages of overcoming the toxæmia and dehydration and the return to more normal metabolism reduce the operative risks and greatly outweigh the risk that spread of growth during this time may be the factor determining ultimate cure. An early and potentially curable case may be lost by inadequate pre-operative preparation.

In few types of malignant disease is pre-operative preparation so important as in cancer of the stomach. In cancer of the breast, pre-operative treatment is

simple and its efficiency or otherwise does little to determine the immediate outcome of the operation. In cancer of the stomach, the position is reversed and the immediate outcome of the operation is often largely determined by the efficiency of the pre-operative preparations. The objects of pre-operative preparation are as follows: (1) To overcome dehydration; (2) To overcome toxæmia; (3) To overcome as far as possible nutritional defects; (4) To overcome anæmia; (5) To check bacterial activity; (6) To overcome distension, atony, and congestion of the stomach.

Personally, I must confess to a feeling of embarrassment in using the word 'toxæmia'. It is a word that is used so freely, so thoughtlessly, and often so unjustifiably that it almost seems to have become debased in the process. But in the case of cancer of the stomach the toxæmia is real and its effects are measurable in many different ways. The details of pre-operative treatment must vary considerably from case to case, depending upon the relative importance of the various factors, and only the essential principles will be referred to here.

Dehydration.—This may be overcome by the giving of large amounts of fluid by the mouth, by the rectum, subcutaneously, or intravenously. Because the patient *can* exist on a markedly reduced fluid intake, we should not assume the existence is a healthy one. The urinary output should of course be watched.

Toxæmia.—This may be largely overcome by the judicious administration of fluids, glucose, and chlorides, but it is essential that the effect of these should be watched by examinations of the blood at least every second day to determine the levels of urea, the chlorides, and the alkali reserve.

Nutritional Defects.—No measures can overcome the weight loss of cancer of the stomach. It may, however, be possible with the aid of the dietitian to devise a diet which is palatable to the patient, which he can take and digest readily, and which contains an adequate number of calories and adequate amounts of protein, fat, carbohydrate, minerals, and vitamins. If we consider for one moment the type of diet which many of these patients have been getting during the weeks or months before admission to hospital, then the necessity for some correction of this before undertaking major surgery becomes apparent. Possibly vitamin C should be given especially liberally. The body should always be given the opportunity to replenish its glycogen reserves.

Anæmia.—No major operation should be carried out on a patient with cancer of the stomach when the hæmoglobin value is less than 70 per cent. Transfusion will probably be necessary in many cases, but, when possible, iron should also be given to provide a reserve supply for use during the post-operative period.

Bacterial Activity.—Patients with cancer of the stomach have ideal conditions for the exhaltation of the bacterial flora of the mouth, œsophagus, stomach, and intestines. And it is this increased bacterial activity which is responsible for so much of the immediate post-operative mortality. The mouth should be kept scrupulously clean and hydrochloric acid administered daily to act as an antiseptic barrier in the stomach and intestines. Gastric lavage also assists this process, both mechanically and by the improvement in gastric secretory function.

Distension.—Operations are still too often carried out on the distended, dilated, and atonic stomach. This distension leads to atony of the gastric muscles and disturbances of blood-supply, and it renders the organ unequal to the reparative processes consequent upon operation. These changes also favour increased bacterial

activity. Marked improvement may follow gastric syphonage and gastric lavage, and, with the improvement resulting from the overcoming of toxæmia, a pylorus which was apparently completely obstructed may again allow food to pass through.

Operative Problems.—The choice of anæsthesia and the technique of the actual operations are matters which must always be largely governed by personal preference and by local conditions. In this country general anæsthesia is the rule, but on the Continent Finsterer has popularized the use of local and splanchnic anæsthesia even for the most extensive resections. There is no doubt that with local anæsthesia the patients stand long operations better than with general anæsthesia.

The operation itself must be either a partial or a complete gastrectomy, but the latter is rarely practised because of the high mortality resulting from the technical difficulties. The type of partial gastrectomy must also be a matter of personal preference rather than of fundamental principle. In this, morbid anatomical factors are of paramount importance, and the site, size, and nature of the growth are the determining ones. The ideal operation must remove the involved portion of the stomach, and on the cardiac side, particularly on the lesser curve, it must include at least 4 cm. of apparently healthy tissue (Verbruggen[11]). Spread to the duodenum is unusual, but it occasionally occurs, as has been shown by Castleman,[12] and 3·0 cm. of this should be removed. The ideal operation must also include removal of the regional lymph-glands, and in this connexion it must be remembered that, although apparently normal glands may contain microscopic deposits, enlarged glands may be the result of infection. Hæmostasis and suturing must be perfect and without tension if infection is to be avoided, and here the lesser curve is the danger-point. Finally, the anastomosis should be of a type unlikely to result in further gastric obstruction from spread of growth.

Growths of the body and of the pyloric portion are within the so-called operable zone. Technically, the former are much easier to deal with than the latter. Growths of the leather-bottle or diffuse type can only be dealt with by means of total gastrectomy, whilst growths of the cardia and the fundus present many of the problems of œsophageal surgery.

Post-operative Problems.—As the immediate post-operative problems do not materially differ from the post-operative problems of other major gastric operations, they will not be discussed here.

PROGNOSIS

The question of prognosis in cancer of the stomach must be considered along three lines in relation to the problem of surgical treatment: first, in terms of the operability rate, secondly in terms of immediate post-operative mortality, and finally in relation to ultimate prognosis.

Operability Rate.—It is almost impossible to obtain accurate data relating to the operability of cancer of the stomach. General hospital data provide the most reliable guide to operability at the time the patient comes into the hands of the expert, but there are obvious fallacies in accepting operability rates from clinics which are largely surgical, or from individual surgeons. However useful these latter sources of data are, they do not provide any exact indication of operability.

In the series of 506 cases already reported, operation was carried out in 351 or 68·4 per cent. This figure includes all types of operation. But the figures

which are of primary importance are those relating to the radical operations of partial or complete gastrectomy. In the above series of 506 cases, partial gastrectomy was carried out in 54, or 10·6 per cent of the total, or in 15·3 per cent of the total cases explored.

Balfour,[13] quoting the figures of the Mayo Clinic for 1920–30, states that resection of the growth was possible in 22·62 per cent of patients with gastric cancer, or in 43·16 per cent of the cases explored.

Walton,[14] referring to his own figures for 230 cases, gives the resection rate as 43·9 per cent.

Post-operative Mortality.—The patient with a cancer of the stomach is usually an extremely poor surgical risk, almost irrespective of the nature of the operation that is carried out. For some patients, therefore, surgery must inevitably be a form of euthanasia. In the present series of cases the following were the main immediate post-operative mortality-rates :—

Operation	*No. of Cases*	*No. of Deaths*	*Mortality*
Partial gastrectomy	54	18	33·3 per cent
Anterior gastro-enterostomy	45	23	51·1 per cent
Posterior gastro-enterostomy	93	30	32·25 per cent
Exploratory laparotomy	136	29	21·3 per cent

Again, the only mortality-rate which is of immediate concern is the rate of gastrectomy, but the other rates have been quoted to show that there is a basic mortality-rate which is independent of the actual type of operation and is determined by the nature of the underlying disease. Even in the cases in which nothing more than exploratory laparotomy was carried out, the mortality-rate reached 21 per cent.

At the Mayo Clinic during the years 1921–30 inclusive, partial gastrectomy for cancer of the stomach was carried out on 1080 persons, with a mortality-rate of 13·05 per cent (Gray, Balfour, and Kirklin[15]).

Walton[14] records a mortality of 28 per cent for radical resections of the stomach for malignant disease.

Ultimate Prognosis.—The low operability rate for cancer of the stomach and the high mortality-rate from gastrectomy leave but a slender remainder on which to base the ultimate surgical cure. The significance of this can best be appreciated if the figures quoted are considered in the following way :—

For each 100 cases of cancer of stomach admitted to hospital,
68 cases of cancer of stomach were explored,
10 cases of cancer of stomach had partial gastrectomy,
7 cases of cancer of stomach survived this radical operation.

Thus, out of every hundred cases admitted there were only seven who left hospital having had an operation which might possibly result in a permanent cure. Unfortunately it has not been possible to follow up all the cases in this series and so complete the story, but the available evidence does not suggest that more than one patient in each hundred survived the five-year period.

Cure of the disease after partial or complete gastrectomy is rare, but at present surgery offers the only known cure. Balfour[13] in 1931 reported 128 ten-year cures. Walton[14] (quoted above), writing in 1936, quoted 20 cures of over five years, or a percentage of 6·1 per cent of all cases coming under his care. Statistics can be multiplied on this point, although accurate comparison between them is almost

impossible. But at least we do know that radical surgery offers the only cure, and that large numbers of five-year cures have been reported, though the cures represent but a small proportion of the total number of cases of cancer of the stomach.

Saltzstein and Sandweiss[10] record some interesting data in this connexion. These writers found that as the result of an analysis of all registered deaths from cancer of the stomach in a population of 1,730,000 over a period of one and a half years, only 3 per cent had had the chance of cure which was represented by recovery from a radical operation.

If the surgeon is seriously to influence the mortality-rates from cancer of the stomach, then he must carry out radical operations in a much larger percentage of cases, and also diminish his post-operative mortality. But successful surgery demands earlier cases, and the surgeon cannot be expected to perform miracles.

FUTURE EDUCATIONAL AND ADMINISTRATIVE PROBLEMS

During the course of this paper I have tried to visualize cancer of the stomach in perspective as a biological problem, as a clinical problem in the community, and as a problem which in the light of our present knowledge can only be dealt with along surgical lines. As far as possible I have concentrated on those aspects which have seemed to me to be closely related to the question of successful surgery. The problem is vast and disquieting, but we must face it honestly and courageously.

Some of the factors in the situation are quite beyond our control, such, for example, as the characteristic vagueness of many of the early symptoms, and the essential morbid anatomical features of the disease. On the other hand, there are many factors which involve the patient, his medical adviser, the surgeon, and the hospital administration which are at least subject to some measure of control. These potentially controllable factors must be so influenced as to obtain earlier diagnosis, increased operability, and diminished post-operative mortality. How can these ends be attained?

The Patient.—The vagueness of onset in many of the cases and the fact that indigestion is the most prominent symptom both deter the patient from taking early medical advice. Confidence in the general practitioner and the recognition that indigestion should not be accepted merely with resignation and fortitude will be of more value than the introduction of periodic overhauls. I do not believe that our results will be improved by driving the patient to the doctor every six or twelve months to see if there are any symptoms or signs of malignant disease. In any case, the early diagnosis of cancer of the stomach is not possible without detailed laboratory investigations. We should at least try to educate the patient to consult his medical adviser for departures from his usual health.

The Medical Adviser.—The problem of the medical adviser is complementary to that of the patient. Confidence must beget confidence. If, in a patient of cancer age, symptoms persist which may possibly be those of early malignant disease, then the patient must be passed on to the expert for complete investigation. Medical education must bear some of the responsibility for the failure to live up to this precept. The overloading of the medical curriculum and the intensive methods employed in undergraduate medical education often diminish a critical outlook, and dull a naturally sceptical turn of mind. A critical mind will suspect that a dyspepsia which does not rapidly respond to simple measures may be due to organic disease which *may* be cancer: an uncritical mind will repeat the alkaline

mixture and the assurances that the condition is only one of 'indigestion', until the growth is no longer operable.

The Surgeon.—Cases of cancer of the stomach are not always referred direct to the surgeon, and although responsibility for the intervening stages of investigation is often taken by the physician, it is the surgeon who must undertake responsibility for all cases in which operation is possible. In this paper I have especially stressed the problems as they confront the surgeon in relation to the radical treatment of the disease, and although those problems remain unsolved I have indicated directions in which progress may be expected. The fact has been stressed that the surgery of the stomach must be undertaken in the knowledge of all the functions of the stomach and not merely in terms of the morbid anatomy of the malignant process. The nutritional aspects of gastric disease are of fundamental importance in connexion with the high post-operative mortality. Success in the surgery of cancer of the stomach must be based on the twin pillars of sound cancer surgery and sound gastric surgery.

Administration.—At the present time, existing hospital accommodation and facilities do not offer the possibility of radical surgery in more than a very small proportion of potentially resectable cases. A more immediate and pressing problem is that of the internal delay which so often still takes place between the patient's first attendance at hospital and his admission to a surgical ward with a diagnosis of cancer of the stomach. We cannot be too critical of the delays occasioned by the patient or his medical adviser if we are merely to add to these delays those due to administrative defects.

For the purposes of this paper I have drawn freely upon the work of many others who have interested themselves in cancer of the stomach, and I am conscious of the debt I owe to all upon whose work, experience, or difficulties I have drawn. In concentrating on the problem of cancer of the stomach, I have had to concentrate largely on those difficulties which seem at times almost to surround the surgeon. I have done this because I believe that in a clinical problem of such a nature it is along these lines that our knowledge may be advanced, and by this means that we as surgeons may do something more than at present to bring hope to those who suffer from one of the most frequent, distressing, and fatal forms of malignant disease.

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DOUBLE GALL-BLADDER

REPORT OF A CASE AND REVIEW OF 38 CASES

By E. STOLKIND

Double gall-bladder is rare, but has a practical interest for the surgeon, physician, and radiologist. This type of gall-bladder has been classified into; (1) Bifid or bilobed (vesica fellea divisa)—i.e., one gall-bladder divided usually by a longitudinal septum into two parts, the transverse section showing two cavities, though occasionally by a transverse septum, the longitudinal section showing two cavities. (2) Accessory gall-bladder or double gall-bladder, i.e., two separate organs, mostly with two separate cystic ducts. The two cystic ducts either unite before emptying into the common bile-duct—Y-shaped type—or empty separately into the common bile-duct—ductular type.

INCIDENCE

In Animals and Birds.—Two and even three or more gall-bladders have been found in domestic and in wild animals. The prevalence of double gall-bladder in domestic animals is at present difficult to determine. Boyden (1926), in America, states that he has found accessory gall-bladders in a considerable number of cats, an average of 1 in 28 in calves, 1 in 85 in sheep, and 1 in 198 in pigs.

My inquiries show that an accessory gall-bladder is considered a rare occurrence in England. For example, Dr. Tom Hare, formerly Professor of Anatomy, Royal Veterinary College, London, in a personal communication states that for many years he has paid particular attention to gall-bladders of animals and birds. His necropsies of domesticated animals as well as of wild animals and birds run into several thousands, but congenital duplication, partial or complete, appeared extremely rarely. He had seen one case in a dog. Acquired deformities of the gall-bladder are not uncommon, especially in herbivorous animals (cattle and sheep); occasional cases are met with in dogs, rabbits, and birds. According to orthodox Jews an animal with an abnormal gall-bladder, e.g., with two gall-bladders, may be unfit for food and therefore the state of this organ has to be investigated. Inspector Herzberg (London) informed me that he has only noted one case of absent gall-bladder, this being in an ox. Between 1909 and 1916 33,386 calves passed through his hands in the orthodox Hebrew slaughter-house; during the four years 1927–30, 9165 oxen and 6234 calves and sheep. He has not found among these a true case of accessory gall-bladder. I had similar information from other slaughterers.

In Human Beings.—The size of an accessory gall-bladder in human beings varies. It may be of normal size or one may be smaller than the other. It may lie either close to the other or the two may be widely separated. One may be embedded within the liver, and the other in its normal fossa. One or both

gall-bladders may be healthy or diseased. I was able to find records, all original, of 38 cases of double gall-bladder found either at necropsy or at operation. These, with our case, make 39 cases in human beings. In addition, in one of our cases, in an infant, the gall-bladder was divided by several septa. The case was that of a girl, aged nine weeks, who had had jaundice since birth. The very small gall-bladder was divided by several septa into chambers filled with clear fluid. There was congenital absence of the hepatic and common bile-ducts, with biliary cirrhosis of the liver.

Similar gall-bladders were reported by Frensdorf (child, aged seven months), Feyrter (child, aged three and a half months), and by Sudo (child, aged three months). There were 21 females and 9 males, and in 9 cases the sex was not stated. Their ages ranged from a few days to 69 years. Seven were children below 11 years of age. In the case of Blasius the age is given as XI in one edition and as ii in another.

Among the 39 cases considered in this paper there are 7 recorded cases of bilobed gall-bladder, 5 with a longitudinal septum and 2 with a transverse septum. According to their descriptions these can be called instances of congenital bilobed gall-bladder (vesica fellea divisa). Our case must be added to these, making 8 in all. In each of these cases there was only one cystic duct.

CASE REPORT

I am indebted to Professor H. M. Turnbull, Institute of Pathology, London Hospital, for this case.

Man, aged 53 years, with no history of previous illness, died from glioma of the brain.

AT AUTOPSY.—On section the gall-bladder was found to be completely healthy, there being no signs of any disease, no cholecystitis, nor stones. The gall-bladder was divided into two equal loculi by a septum containing a narrow orifice, 2 cm. in circumference, uniting the two healthy cavities. As the gall-bladder was quite healthy, the septum was definitely of congenital origin, due to a defect of development.

DIAGNOSIS

The earliest mention of double gall-bladder which I have been able to find is that of Pliny, who wrote that a double gall-bladder had been found in a victim sacrificed by Augustus. A true diagnosis of a bilobed or an accessory gall-bladder is usually difficult, as there are no signs or symptoms typical for this congenital abnormality. Almost all reported cases were diagnosed either at operation (24 cases) or at the necropsy. The operation has generally been performed because of cholecystitis or cholelithiasis. X-ray examination may assist in some cases, e.g., in Hilt's case the radiograph showed that the gall-bladder appeared to be divided into two parts. In Nichol's case X-ray examination revealed two distinct rows of stones in the gall-bladder region. In all other reported cases in which a double gall-bladder has been found either at the operation or necropsy, it was not visualized. Contrary to these cases, there are reports of double gall-bladder seen only at X-ray examination, e.g., cases of Braunschweig (double gall-bladder with two cystic ducts); Cave (two shadows in each of two females); Hayes (two gall-bladders with two cystic ducts); Climan (double gall-bladder); Blaine (two distinct shadows overlapping each other); Weiss; Corachan, Figueras, and Faixat (shadows of

two gall-bladders with one cystic duct). In none of these cases was the X-ray diagnosis later confirmed at an operation or necropsy. Syphilitic and other pathological changes in the gall-bladder—diverticula, congenital furrow only on the wall of a normal gall-bladder, as in one of my cases shown at the Royal Society of Medicine (London), or Phrygian caps—should be borne in mind when diagnosing congenital bilobed or double gall-bladder by X-ray examination.

The diagnosis of bilobed gall-bladder is sometimes difficult even at the examination of the removed specimen, e.g., in cases where there is chronic cholecystitis. In such cases the gall-bladder may be divided by a septum or septa into two, three, or more compartments. The septa are generally incomplete and usually transverse. Several cases recorded as bilobed gall-bladders have been hour-glass gall-bladders with pronounced pathological changes, e.g., the cases of Morton; Hinder; Deaver and Ashhurst; McMahon; Ugelli; Esau; the second case of Phillips, Isaac, and MacDonald; and that of Huard, Ho-Dac-Di, and Ton-That-Tung. In Ullmann's case, as well as in that of Otto, there was a diverticulum of the gall-bladder. Judd, McIndoe, and Marshall did not report their own case of a double gall-bladder as Gross states. All these cases are not included in the following series, as I considered them not proven. The case of De Graaf which Holderman wrote of was an ox with a double gall-bladder. I have not included Gohl's case as I did not find it in *Comm. liter. rei. med. Norimb.*, 1738, the reference given by Haller and copied by others.

SUMMARY OF REPORTED CASES

Particulars of all the proved cases of double gall-bladder, including the case reported by the author, are given below.

Table I.—Cases of Congenital Bilobed Gall-bladder

Case	Author	Sex	Age	Symptoms	Findings
1	Cruveilhier	—	—	—	Gall-bladder with longitudinal septum dividing it into two parts to the neck, which was single. Two cystic ducts, one emptying into hepatic and the other into common bile-duct
2	Rycroft	F.	42	Attacks of biliary colic for 13 years	Operation; longitudinal septum dividing gall-bladder into two unequal loculi not extending to the neck; stone in the smaller loculus
3	Meyer	F.	55	Attacks of biliary colic for many years	Operation; inflamed thickened gall-bladder with stones; longitudinal septum; one cystic duct
4	Phillips, Isaac, and MacDonald	M.	40	Colic	Two operations, the second being cholecystogastrostomy; two unequal gall-bladders; larger buried in the mass of adhesions; common ampulla and single cystic duct
5	Hilt	F.	30	Pain in abdomen; radiograph, gall-bladder divided into two parts	Partial cholecystectomy; inflamed gall-bladder; fundus divided into two equal lobes; one cystic duct

Table I.—Cases of Congenital Bilobed Gall-bladder—*continued*

Case	Author	Sex	Age	Symptoms	Findings
6	Dévé	—	1	—	Circular superficial constriction on external part of the gall-bladder near fundus; transverse septum divided it into two unequal cavities with normal walls
7	Stadnizky	M.	42	Dysentery	Gall-bladder healthy; circular constriction on its surface; transverse septum dividing it into two unequal cavities with normal walls
8	Stolkind	M.	53	Tumour of the brain and purulent bronchitis	Completely healthy gall-bladder divided by a septum into two cavities

Table II.—Cases of Accessory Gall-bladder

Case	Author	Sex	Age	Symptoms	Findings
9	Blasius	M.	2 (? XI)	—	One normal gall-bladder and near it another, smaller and finer; two cystic ducts
10	Witty. (This case was wrongly attributed to Foot)	F.	31	Indigestion with vomiting; stone in left kidney	Much enlarged liver with adhesions; two gall-bladders separated one from another by the length of a hand, left smaller; stone in left kidney; phthisis of the lungs
11	Huber	—	Few days	—	Two distinct gall-bladders, each with its fundus, neck, and cystic duct; right larger than the left; on the left there was a slender enlargement
12	Purser	F.	11	Died from scarlet fever	Two separate gall-bladders; two cystic ducts; three hepatic ducts
13	King's College Hospital	—	—	—	Two separate normal gall-bladders, both open at their necks. (*Specimen*)
14	Sherren	F.	25	Attacks of colicky pain in right hypochondrium	Two separate gall-bladders with two cystic ducts; operation
15	Schulz	M.	35	Cholecystitis and cholangitis	Large gall-bladder divided into two equal parts, each with stones; cystic duct double for half its length, polydactylism; operation
16	Schachner	F.	52	Gall-stone colic for more than fifteen years	Operation; double gall-bladder, each with an independent neck and apparently two cystic ducts; stones
17	Haberer	F.	Adult	—	Operation; gall-bladder and cystic duct divided by a longitudinal septum; stones
18	Wischnevsky	F.	31	Fever, icterus, and palpable tumour	Operation; two gall-bladders with two cystic ducts; accessory at some distance from the other and containing pus; stones in left cystic duct

Table II.—CASES OF ACCESSORY GALL-BLADDER—*continued*

CASE	AUTHOR	SEX	AGE	SYMPTOMS	FINDINGS
19	Braun	—	—	—	Operation; one gall-bladder with adhesions and stones was removed; the accessory and its duct not removed.
20	Nichols	M.	51	Radiograph; two distinct rows of stones	Two separate gall-bladders each with stones and two cystic ducts; operation
21	Wakeley	F.	56	Radiograph; gall-stones	Smaller gall-bladder contained stones; two cystic ducts; operation
22	Garofalo	F.	37	Attacks of colicky pain for fifteen years	Accessory gall-bladder small; changes typical of hydropsy; no stones; two cystic ducts; operation
23	Priesel	M.	54	Peritonitis	Superficial furrow on the lower surface of the gall-bladder which was divided by a longitudinal septum into two parts; two cystic ducts; operation
24	Wolfson	F.	19	Attacks of pain in right hypochondrium for nine years; radiography, stones	Accessory rudimentary gall-bladder arising from common duct with two stones; other gall-bladder normal; cholecystectomy
25	Petrina	F.	—	Abortion	Gall-bladder divided into two by septum; two cystic ducts; stone in one cystic duct; operation
26	Holderman	F.	30	Attacks of pain in right hypochondrium for seven years; radiograph, stones	Gall-bladder with sagittal septum, stones in each chamber; two cystic ducts; operation
27	Minin	F.	43	Twelve pregnancies and frequent attacks of colicky pain in right hypochondrium; jaundice	Operation; fundus consisted of two cupolas; furrow on surface; longitudinal septum divided enlarged and adherent gall-bladder into two parts down to neck (which had two openings) and to cystic duct; stones in each chamber
28	Croudace	F.	52	Attacks of colicky pain and tenderness in right hypochondrium; radiograph, suggestive of cholecystitis	Accessory gall-bladder half-size and containing stones; two cystic ducts, that of second gall-bladder penetrated into liver; operation
29	Strukov	M.	$1\frac{2}{12}$	Died of scarlet fever	Two normal gall-bladders with two cystic ducts opening separately into common bile-duct
30	Kennon	M.	69	Acute suppurative cholecystitis and stones	The small gall-bladder had a scarred fundus from first operation; each gall-bladder opened separately into common bile-duct, which contained stones; cholecystectomy
31	Slaughter and Trout	F.	32	Frequent attacks of pain in right hypochondrium; radiograph negative	Operation; accessory gall-bladder atrophic and buried in liver; cystic duct from accessory gall-bladder joined other duct before it entered common duct

Table II.—CASES OF ACCESSORY GALL-BLADDER—*continued*

CASE	AUTHOR	SEX	AGE	SYMPTOMS	FINDINGS
32	Herrmann, Hicks, and Martin	F.	42	Epigastric distress, jaundice, and pruritus	Operation; two separate cavities in gall-bladder with stones in each; two cystic ducts
33	Stohr	F.	19	Colicky attacks	Operation; two gall-bladders with one small cystic duct; adhesions
34	Gross	F.	$3\frac{3}{12}$	Bronchiectasis and broncho-pneumonia	Accessory gall-bladder partly buried in liver; two cystic ducts
35	Wilson	F.	55	Attacks of pain for twenty years	Operation; gall-bladder divided by longitudinal septum into two cavities; two cystic ducts; papilloma of gall-bladder; acute and chronic cholecystitis, cholelithiasis, empyema

The last 4 of the 39 cases are insufficiently described.

Case 36 (Porter).—Girl, aged 5 years; accessory gall-bladder connected by a small stem to the normal one. The author was unable to give me any further particulars in this case. (Boyden mentions three unpublished cases in letters to him. They were all insufficiently described.)

Case 37 (Hardesty).—A small accessory gall-bladder was given off from the cystic duct.

Case 38 (Erdmann).—A double gall-bladder with a single cystic duct.

Case 39 (Lambert).—Small accessory gall-bladder, being an outgrowth of the common duct.

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SUBMARINE SALVAGE

THE AIR IN A SUBMERGED SUBMARINE: MEANS OF EXIT WHEN SUBMERGED: AND DISABILITIES OF THE SURVIVORS

By Surgeon Lieutenant-Commander S. JENKINSON, Royal Navy

By this that stranger knight in presence came,
And goodly salved them. (Spenser.)

INTRODUCTION

It became evident with the dawn of the twentieth century that submarines were of utility to a navy, both for offence and for defence.

With their arrival the determination of the changes in the atmosphere of a sealed compartment, due to the respiration of human beings, was required in respect of the production and the percentages of deleterious gases, and the increase of air pressure in the compartment in certain circumstances. For if a submarine is sunk, water as it enters will compress the air in the submarine until the air pressure equals that of the depth of water. At 33 ft., which is + 1 atmosphere, the air inside will be compressed to half its original volume; and at + 2, 3, or 4 atmospheres or 66, 99, or 132 ft. the air volume will diminish in accordance with the air pressure.

The air of a sealed compartment containing human beings will become abnormal in that there are:—

1. Chemical changes:
   a. Increase in carbon dioxide;
   b. Diminution of oxygen.
2. Physical changes:
   a. Increase of temperature;
   b. Increase of moisture;
   c. Vitiation due to emanations from the inmates.

The installation of chemical absorbents used to be adopted to recondition the air in submarines. Soda lime was carried to absorb carbon dioxide and water, but this method was discontinued after trials carried out in the worst heat conditions in the Mediterranean had shown that, when a submarine cruises on the sea surface, the oxygen content of the air rarely falls below 20 per cent and the carbon dioxide rarely exceeds 1 per cent, and after having been submerged for twenty-four hours there was not more than 3 per cent of carbon dioxide in the air, which was not sufficient to affect the crew adversely. It was reckoned that thirty-six hours would elapse before the carbon dioxide percentage would rise to 5, and it was considered that the factors which determined the noxious properties of the air under such conditions were physical and not chemical, the percentage of carbon dioxide merely providing an index of air impurity. Since then no absorbent chemical apparatus has been carried. Instead increased convection of air is maintained by electric fan systems to promote evaporation from the lungs and skin.

But if, when submerged, the submarine cannot be brought to the surface, the men must within a certain lapse of time ascend from the wreck to the sea surface. To enable men to do so without likelihood of drowning, a respiratory apparatus is worn from which oxygen is breathed during the process of escape.

The procedure of escape may only be adopted when the submarine is lying in comparatively shallow water. Up to 300 ft., which is the limit for survival, the situation of the crew will be hazardous. In avoiding one of the complications they may incur another. They may have to choose whether to contract carbon dioxide poisoning, oxygen poisoning, chlorine poisoning, or compressed-air illness, or risk exposure, and may eventually suffer several of these maladies. Their choice will be dependent on such data as the depth of the submarine causing an increased degree of air pressure when about to escape, and whether rescue is at hand at the sea surface, for until help arrives they may decide to remain below.

In order that the several disabilities may be explained it is convenient to consider :—

1. The physiology and respiration of the blood gases.
2. Chlorine gas poisoning.
3. Compressed-air illness.
4. Diagnosis.
5. Differential diagnosis.
6. Methods of escape.
7. The escape apparatus.
8. Treatment of casualties.

1. PHYSIOLOGY AND RESPIRATION OF THE BLOOD GASES

a. Carbon Dioxide Poisoning.—

Inspired air contains 0·04 per cent carbon dioxide
Expired air contains 4·1 per cent carbon dioxide

The rise in percentage of carbon dioxide in a submarine will increase in ratio to the number of men and the time remaining below the surface. Given the cubic air capacity in the submarine, the percentage can be calculated.

Up to 2 per cent of carbon dioxide there are no noticeable effects while the submarine remains sealed.

At 3 per cent there will be hyperpnœa.
At 4 per cent there is discomfort when working.
At 5 per cent hard work is impossible.
At 6 per cent there is panting.
At 10 per cent distress is evident.
At 12 per cent coma will supervene.
At 20 per cent death occurs.

Carbon dioxide stimulates the respiratory centre and at 2 per cent there is an increase of respiration by 30 per cent and lung ventilation by 50 per cent, the alveolar content of the air remaining constant within certain limits. But this limit is exceeded when, with reference to the laws of gases in liquids, the blood will take up twice as much carbon dioxide if the pressure is doubled.

The volume of carbon dioxide that the blood can absorb is due to the fact that while only three volumes are carried in physical solution, 55 volumes are

carried by combining with the base in the plasma to form a bicarbonate. This union can be expelled by acid or a vacuum, so proving the data.

Moreover, deoxygenated blood, such as is found in the inmates of a submarine after submergence, has a greater carrying capacity for carbon dioxide. Thus at 2 atmospheres or 33 ft. depth, 1 per cent carbon dioxide has the same effect as 2 per cent at the surface, or as ½ per cent at 99 ft. or 4 atmospheres; the greater the depth the greater is the effect of the percentage of carbon dioxide. (*Fig.* 560.)

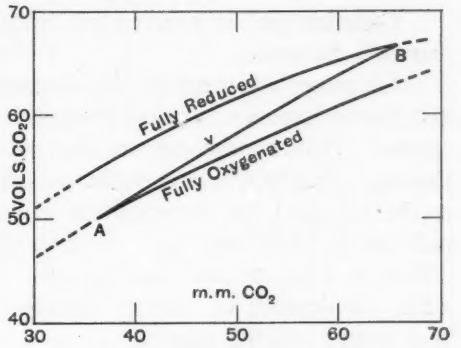

FIG. 560.—CO_2-dissociation curves of fully reduced and fully oxygenated blood. AVB is the physiological curve, along which the CO_2 dissociation operates in the body. (*Redrawn from Christiansen, Douglas, and Haldane.*)

If this percentage were 4 per cent at 1 atmosphere, which is the constant pressure while the submarine is sealed, the tension and therefore the relative percentage will be doubled to 8 per cent, or quadrupled to 16 per cent, when attempts are made to equalize the water pressure by opening the hatch at 33 ft. and 99 ft. respectively.

b. **Lack of Oxygen.**—As the carbon dioxide percentage rises there will also be diminution of oxygen, which is normally 21 per cent in inspired air and 16 per cent in expired air. It is found that when the percentage of oxygen diminishes to 15 per cent there is increased pulmonary ventilation and at 13 per cent collapse is imminent. Respiration is not affected by increase of tension of oxygen, as in the case of carbon dioxide, but diminution of tension below 13 per cent will result in apnœa.

c. **Oxygen Poisoning.**—At an air pressure higher than normal atmospheric pressure, oxygen is taken up by the blood to such an extent that the oxyhæmoglobin is not reduced, which, retaining the base normally available, prohibits the elimination of carbon dioxide from the tissues. Hence there is a rise in the percentage of carbon dioxide in the tissues. Twitchings and convulsions of the head and neck result—the symptoms of oxygen poisoning.

Oxygen may be respired at 1 atmosphere for several hours without ill effects; but at 4 atmospheres pressure will cause convulsions, and at 10 atmospheres or 300 ft. depth, is rapidly fatal owing to paralysis of the respiratory centres, the only premonitions being twitchings of the facial muscles and paræsthesia at the extremities.

Wearing the oxygen escape apparatus, which contains 75 per cent of oxygen and 25 per cent nitrogen (which has accumulated from the blood), it is unlikely that convulsions will appear in less than the following times:—

Depth of Water (in ft.)	*Minutes*
100	60
125	20
150	8
200	6
250	4
300	3

(Damant.)

Thus the respiration of oxygen at a very high pressure will cause direct death to the tissues. Oxygen respired for hours at normal pressure may bring on consolidation of the lungs.

2. CHLORINE POISONING

Chlorine gas is evolved by electrolysis from access of sea water to the submarine's batteries.

Chlorine is irritant to the respiratory tract and causes paroxysms of coughing and frothy expectoration. Either pallor and collapse or cyanosis and dyspnœa will appear; both syndromes are due to a lack of oxygen subsequent to inflammatory changes and pulmonary œdema. It is obvious that swelling of the alveolar epithelium and the interposition of œdema will inhibit gaseous exchange between pulmonary blood and air. Carbon dioxide, being more soluble than oxygen, will diffuse at a higher rate than oxygen. Thus absorption of oxygen will be diminished while elimination of carbon dioxide will not be affected, so that pallor, collapse, and apnœa will be evident.

At a further stage, with massive pulmonary collapse, elimination of carbon dioxide will be impeded; the case will then exhibit both shortage of oxygen and retention of carbon dioxide, manifested clinically by blue asphyxia and hyperpnœa, the venous congestion inducing heart failure as a final scene.

3. COMPRESSED-AIR ILLNESS

Approximate composition of air { Oxygen, 21 per cent / Nitrogen, 79 per cent

When a man has breathed air at several atmospheres pressure and then is transferred to normal absolute atmospheric pressure he may suffer compressed-air illness. The symptoms and signs are due to the liberation of bubbles of nitrogen from the blood into the tissues—just as bubbles rise from a soda-water bottle when the pressure therein is released; the water appears natural while the pressure is maintained, but when the pressure is reduced bubbles appear from the liquid. Nitrogen is the only gas which accumulates so in abnormal quantities when a man is subjected to air pressure.

While the men in a submarine are under pressure—that is, while the compartment is being flooded so as to escape—the nitrogen under pressure is absorbed into the blood-stream, and when he ascends to the sea surface on escaping the nitrogen gradually bubbles off, the blood being viscous and therefore slower to give off bubbles when compared with water. If the survivor has been subjected to pressure for any length of time the bubbles will cause serious symptoms. The tissues as well as the blood have become saturated with nitrogen, especially the fat and nerve tissues, at a rate dependent upon their blood-supply. The brain and the cord are quickly saturated, since the circulation is free, while ligaments and joints, with a meagre circulation, are slowly saturated. The converse is true—those tissues which saturate slowly, desaturate slowly, and upon the tissue may depend the time of onset of the symptoms; a lesion of the brain is apparent in a few minutes after surfacing, while if in the joints it may be several hours before being observed. Post mortem the bubbles are most easily seen in the mesenteric vessels. In the spinal cord the bubbles are of just naked-eye visibility, while in the brain they are seldom seen.

In rapidly fatal cases the right heart is distended with bubbles, the pulmonary vessels contain froth, the left ventricle being nearly empty. Preceding dyspnœa is an alarming symptom. It will be realized how important it is that the submarine compartment or chamber should be quickly flooded in order to minimize the time under air pressure.

4. DIAGNOSIS

The symptoms and signs of carbon dioxide poisoning, oxygen poisoning, chlorine poisoning, and compressed-air illness have been in part considered. It remains to amplify the clinical picture.

Carbon Dioxide Poisoning.—As has been remarked under chlorine poisoning, excess of carbon dioxide, with lack of oxygen in the blood, gives a picture of purplish cyanosis, hypernœa, and a bounding pulse.

Oxygen deficiency with normal carbon dioxide content will provide cyanosis of a pale lavender colour, with the breathing rapid and shallow, and the pulse thready.

Oxygen Poisoning.—It has been shown by experiment that the higher the alveolar carbon-dioxide tension the shorter the exposure to oxygen necessary to induce symptoms of oxygen poisoning.

Carbon dioxide tensions are innocuous when associated with oxygen pressures of less than 1 atmosphere, but prove highly toxic when associated with oxygen at 4 atmospheres pressure.

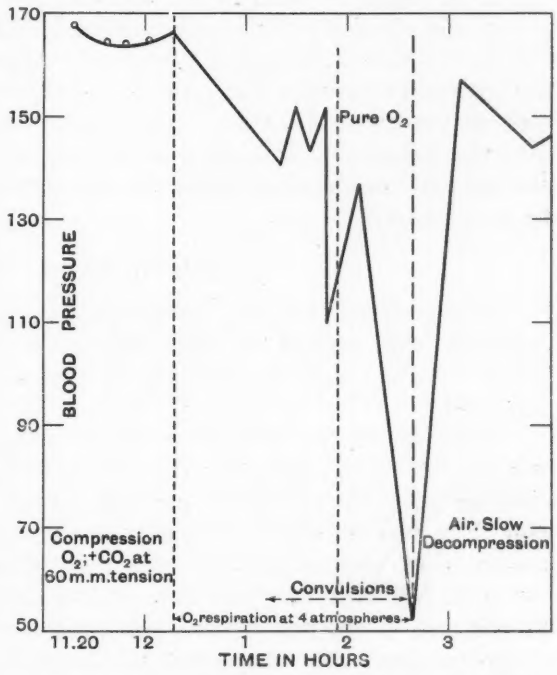

FIG. 561.—Blood-pressure taken during inspiration of oxygen at 4 atmospheres pressure combined with a high carbon dioxide tension in the lungs.

Leonard Hill (1933) observed that when monkeys were first exposed to 5 per cent carbon dioxide and 95 per cent oxygen, followed by an increasing oxygen pressure, convulsions were induced more quickly or at lower oxygen pressures than in animals exposed to oxygen alone. He concluded that the increase of carbon dioxide in the tissues is a factor in the production of convulsions which follow exposures to high pressures of oxygen.

Behnke, Shaw, Shilling, Thomson, and Merser (1934) have shown from their experimental results that it may be inferred that carbon dioxide tensions which would be entirely innocuous when associated with normal oxygen tensions, might prove toxic with an oxygen pressure of 4 atmospheres absolute (*Fig.* 561).

Compressed-air Illness.—Compressed-air illness is manifested in divers ways. Not only the type, but also the time of onset, is variable. It is convenient to classify casualties into :—

1. '*Bends*.'—This is manifested by pains in the joints, or loss of power in the limbs. The pain is severe and the limb is flexed, hence the colloquial term. The bubble of nitrogen is situated in the cartilage or areolar tissue. The knee, elbow, and those joints which have been used in normal work, are commonly affected.

2. *The Skin*.—Mottling and discoloured patches in the skin, colloquially known as 'sandboy's itch'.

3. *Respiratory*.—A sense of constriction in the chest and dyspnœa, known as the 'chokes'.

4. *Nervous*.—Nystagmus, diplopia, giddiness, aphasia, deafness, blindness, convulsions, coma.

5. *Paralytic*.—Hemiplegia, monoplegia, retention of urine.

6. *Alimentary*.—Pain in the stomach and vomiting.

7. *Fulminating*.—Numerous bubbles in the heart and lungs.

8. *Aural*.—Sensations of pressure, deafness, and pain arise in the ear, due to the inequality between the pressure in the tympanic cavity, and are relieved by opening the Eustachian tube. This can be done by swallowing or forcibly expiring with the mouth shut and the nose held tightly.

In view of their variations the symptoms of compressed-air illness may not be recognized.

5. DIFFERENTIAL DIAGNOSIS

When survivors from a submerged submarine appear at the sea surface their condition will depend on their individual resistence; the depth at which the vessel is lying; and the intensity of gaseous pressure to which they have been subjected.

Since for every 2·2 ft. there is 1 lb. pressure to the square inch, the pressure at 130 ft. is 60 lb., when the chamber is adequately flooded to equalize the water pressure. At that pressure 2 per cent carbon dioxide will be equivalent to 12 per cent at normal barometric pressure; oxygen may be breathed from the respiratory apparatus with safety for not more than fifteen minutes.

Thus the survivors may have carbon dioxide poisoning if they have breathed 12 per cent of the gas, oxygen poisoning if oxygen has been breathed more than fifteen minutes, and compressed-air illness if exposed to air pressure for a time, followed by a rapid ascent to the sea surface.

Since treatment is different it is necessary to bear in mind certain distinguishing points: Carbon dioxide poisoning is indicated by blue asphyxia, oxygen deficiency by white asphyxia, chlorine poisoning by coughing and frothy expectoration.

The main means of differentiation of compressed-air illness and oxygen poisoning lies in the time of the onset of the symptoms. Whereas oxygen poisoning will be evident by twitching of the muscles and limbs, convulsions, and coma immediately on reaching the surface, with evidence that the breathing apparatus has been used, on the other hand the sufferer from compressed-air illness may be able to give a history as to the time of exposure to air pressure in the submarine and the time of respiration of oxygen. This 'lucid interval', which varies from ten minutes to four hours before the onset of symptoms, is thus valuable in deciding the treatment of other men in coma.

Of the two diseases, while oxygen poisoning is more probable in escape from a chamber of a modern submarine, because the chamber can be rapidly flooded,

compressed-air illness is more probable if the flooding has taken place in a compartment, when the men will be subjected to air pressure over a longer period of time.

6. METHODS OF ESCAPE.

We shall assume that every available method has been utilized to attract vessels to the scene where the submarine is lying. The method of escape differs with the class of submarine.

***a.* Compartment.**—The initial device, still in use in earlier submarines, was the fitting to a spring hatch, normally shut and secured by nuts, of a twill trunk, packed underneath the hatch, thus stowed ready for use. When utilized for escape of the crew, the trunk may be unfolded, pulled down, and secured firmly by roping to the deck, so that the lower end is about 3 ft. from the deck. (*Fig.* 562.)

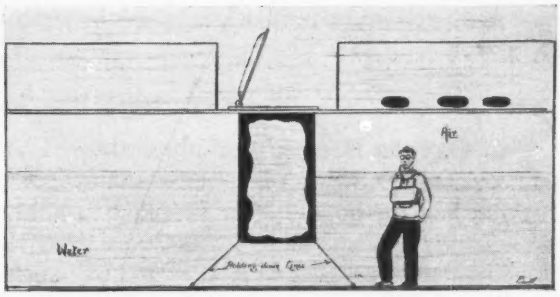

FIG. 562.—Method of escape from submarine compartment.

At the arranged time the submarine compartment is flooded with all dispatch until the internal pressure balances the sea pressure, when the hatch can be opened; the air in the trunk will then vent and the trunk will fill with water. Just before the hatch is opened the men will put in the mouthpiece of the escape apparatus and start breathing oxygen from the bag of their apparatus. They will stand around the twill trunk, and in turn bend down and enter it and escape through the hatch to the sea surface.

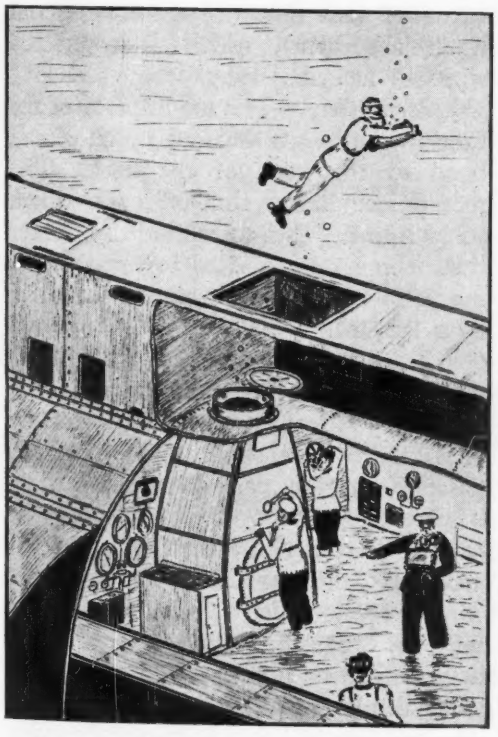

FIG. 563.—Escape from submarine chamber.

***b.* Escape Chamber.**—Submarines of more modern construction (since about 1930) have two escape chambers fitted, one fore and one aft. Each consists of a small chamber tested to a pressure of 75 lb. per square inch and situated in the main gangway. The chamber has a circular watertight door opening to either adjacent compartment; controls for the escaping are in triplicate, and can be worked from either adjacent compartment or from the inside of the chamber. (*Fig.* 563.)

The hatch of the chamber is spring balanced. Two men at a time enter the chamber and shut the door and

begin breathing from their escape apparatus as the water rises. When the chamber is flooded the men inside push the hatch open and escape.

Their movements are seen from the adjacent compartment. The chamber is drained and the procedure repeated, the next two men entering.

The escape chamber is advantageous in that the men waiting to escape are in a dry compartment, and the time submerged and under pressure is not more than about three minutes, whereas a longer period would be required to flood a compartment. Hence there is little danger of oxygen poisoning or exposure due to cold.

A disadvantage is that a man might jamb the hatch, when the chamber would be out of action.

7. THE ESCAPE APPARATUS

a. **Oxygen Breathing Apparatus.**—The Davis Submerged Escape Apparatus (abbreviated, D.S.E.A.) is a self-contained oxygen breathing apparatus which will last for half an hour. It is supplied to submarines in order that the crews may safely rise from the submarine to the sea surface. (*Fig.* 564.)

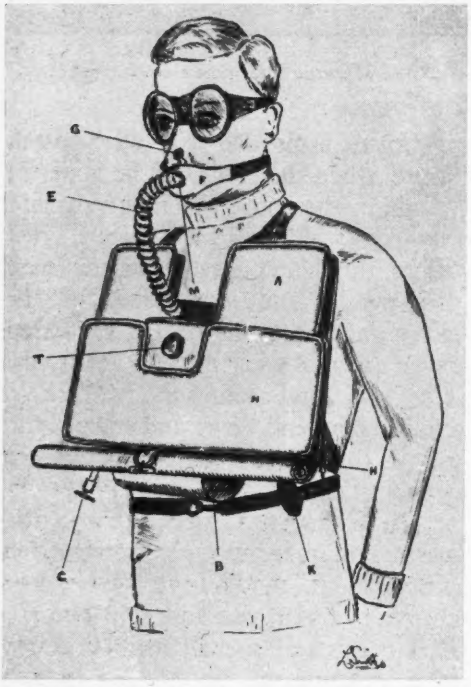

FIG. 564.—A, Horn of breathing bag; B, Oxygen flask; C, Regulating valve; E, Flexible tube; F, Mouthpiece; G, Noseclip; H, Valve for manifold; K, Exhaust valve; N, Breathing bag.

The D.S.E.A. may also be used as a shallow diving dress and as a gas mask.

Breathing Bag.—The set consists of a rubber breathing bag, worn on the chest and secured to the waist by straps. Inside the bag is a canister containing 15 oz. of soda lime (carbon dioxide and water absorbent), which is connected with the facepiece tube.

Mouthpiece.—The mouthpiece of the tube to the bag is secured by an elastic band to the back of the neck. The mouthpiece has a shut-off cock which sticks into the chin to remind the wearer that it must be opened to breathe. A nose-clip is attached to the mouthpiece by a lanyard.

The Flask.—The oxygen flask lies in the pocket below the breathing bag. It contains 56 litres of oxygen at a pressure of 1800 lb. per square inch. Oxygen passes from the flask to the breathing bag, regulated by a valve which may be opened or shut half a turn as required.

Accessory Flasks.—In addition to the main flask, there are inside the breathing bag two miniature oxygen flasks, with soft copper stems which screw into the main charging connexions. If the main supply fails or is exhausted, by manually breaking the copper stems emergency oxygen to last six minutes is released into the bag.

Auxiliary Oxygen.—The breathing bag may also be inflated from a large oxygen cylinder in the submarine by connecting up the valve on its left side, thus conserving the oxygen flask supply until it is required.

Exhaust Valve.—An exhaust valve, placed at the bottom of the bag, consists of a shut-off cock. By means of this valve excess oxygen pressure in the bag is prevented. It must be open when in the water, since rising to the surface the pressure on the bag will diminish, causing expansion and pressure on the lungs, and shut on arrival at the surface, to preserve the buoyancy.

Goggles.—Goggles protect the eyes from oil and keep the vision clear while under water.

Buoyancy Bag.—A buoyancy bag inflated by the breakage of an oxylet small cylinder will bring an unconscious man to the surface whose set is out of action. An apron below the bag should be unrolled and held out by the survivor to slow the ascent to the surface.

***b*. The Drill for the Escape Apparatus.—**

i. When an emergency appears the D.S.E.A. is put on in the ' ready position '.

ii. The compartment or escape chamber is flooded.

iii. If the air is fresh, flooding taking place soon after the accident, the air of the com, artment should be breathed while flooding, but if the air is foul, oxygen from the bag which has been charged from the manifold cylinder should be breathed.

iv. When the water is rising the mouthpiece is inserted, the wearer exhales, puts on the nose-clip, opens the mouthpiece cock, and breathes from the D.S.E.A. oxygen.

Before leaving the submarine the exhaust valve is opened and the buoyancy bag inflated by an oxylet, and the apron is cleared. When ascending through the water the wearer exhales to prevent distension or rupture of the lungs.

On arrival at the sea surface the exhaust valve and mouthpiece cock are shut, the nose-clip taken off, and the bag inflated by blowing into it, the mouthpiece cock being opened and shut synchronously. He lies back and awaits rescue.

After, if there is no oxygen poisoning but a probability of compressed-air illness, oxygen is breathed from the apparatus.

8. TREATMENT OF CASUALTIES

It is assumed that the salvage ship will have arrived at the scene of the submerged submarine; there will have been preparation for the reception of casualties; a casualty station will have been arranged complete with bedding, and facilities for the supply of warmth; the decompression chamber will have been prepared (*Figs.* 565, 566), the resuscitation apparatus of oxygen 93 per cent and carbon dioxide 7 per cent available; the oxygen breathing apparatus in readiness.

A conscious casualty will have been questioned and a history obtained; if the indications are that the survivors have oxygen poisoning they will be treated for shock, convulsions being controlled by morphia.

For cases of collapse due to carbon dioxide, oxygen poisoning, or chlorine poisoning, artificial respiration will be required, Schafer's method being supplemented by inhalations from the resuscitation apparatus.

A man who has suffered from carbon-dioxide or oxygen poisoning at a depth is relieved on arrival at the surface owing to diminution of pressure and fall in the tension of carbon dioxide or oxygen in the blood.

Compressed-air Illness.—Survivors with compressed-air illness would be put into the decompression chamber as soon as possible. An oxygen breathing set would be put on and oxygen breathed for about ten minutes to facilitate

the expulsion of nitrogen bubbles, if there was no indication in the survivors of pre-existing oxygen poisoning. Exercise, massage, and movements would be carried out, the men would then be recompressed until the symptoms were relieved, and decompressed according to the scale from the *Diving Manual*.

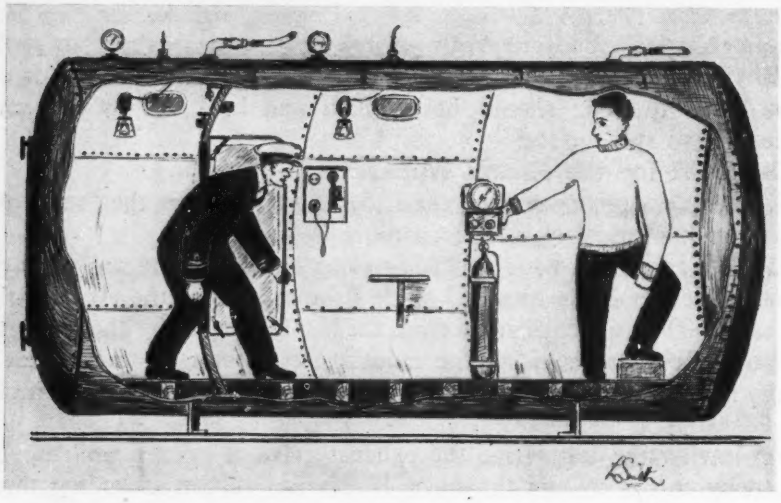

FIG. 565.—Shows survivor exercising in chamber during recompression and decompression.

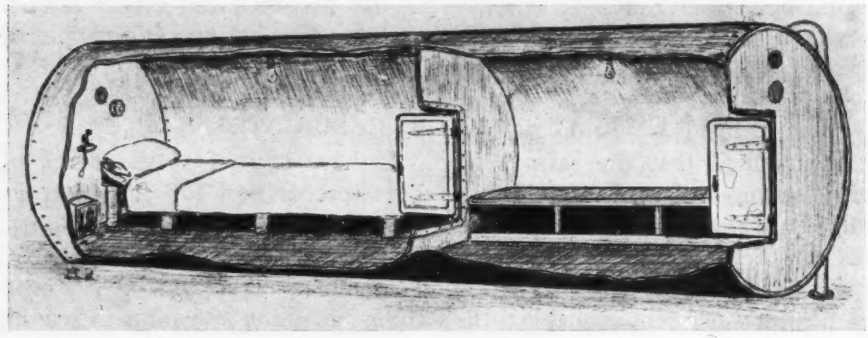

FIG. 566.—A decompression chamber.

Recompression.—Men can be recompressed in any of the following :—

1. A recompression chamber such as is fitted in a diving or submarine depot ship.

2. A ship's disinfector which will contain two men ; possibly in new construction a combined recompression chamber and disinfector will be fitted.

3. In a submarine escape chamber. Two men in each chamber may be compressed up to 75 lb. pressure.

4. By sending a survivor down in a diving suit.

5. In a cold ship's boiler.

Standing Rules for a Recompression Chamber.—

1. The chamber should be available at the scene of the sunk submarine.

2. The chamber should be used immediately and ruthlessly; when a man is in a coma, certain justifiable risks may be taken in accordance with the diagnosis.

3. The chamber should have been tested to guarantee working order. Any leakage from the door should have been repaired and one should be conversant with the means of air supply.

4. The chamber should be available until four hours after the appearance of the last survivor.

5. Gear to be kept in the chamber: bedding, mattresses, blankets, towels, torch, pad and pencil, table of decompression times, watch or clock, thermos flask.

Practical Rules for the Decompression Chamber.—A diver with a knowledge of pressures and compressed-air illness may arrange the bedding, towels, hot-water bottles, and time keeping. It is convenient for him to go into the chamber with the casualties, if space permits. Recompression squeezes the nitrogen bubbles and propels them from the tissues back into the blood-stream, to be expired in the pulmonary circulation.

The survivors are put into the chamber as quickly as possible, being inspected through the glass frame and told by telephone to say when the pain 'lifts'. The symptoms often subside at a pressure of 5–10 lb., and an unconscious man may sit up at 20 lb. pressure.

The paralytic cases are the most obstinate, and up to 100 lb. pressure may be necessary. If relief is not then evident, higher pressure is of no avail. The effective pressure has no relation to the depth at which the diver has been working.

When admitting air into the chamber the first few pounds are run up quickly and then lowered so that the point at which relief occurs may not be overshot. The patients are told to exercise and are massaged until the pain ceases.

When the Pressure per square inch in the Chamber is	*Pressure should fall at*
Between 100 and 90 lb.	4 lb. in 1 minute
Between 90 and 75 lb.	2 lb. in 1 minute
Between 75 and 60 lb.	1 lb. in 1 minute
Between 60 and 45 lb.	1 lb. in 1½ minutes
Between 45 and 30 lb.	1 lb. in 3 minutes
Between 30 and 15 lb.	1 lb. in 5 minutes
Between 15 and 0 lb.	1 lb. in 8 minutes

If the patient shows signs of recurrence while being decompressed, pressure should be raised cautiously until the symptoms disappear, and then decompression restarted at a slower rate as per the *Diving Manual*.

Prophylaxis.—Prophylaxis involves a twofold problem: firstly, the measures to be adopted to avoid mishap to a submarine; and, secondly, the procedure when the submarine is sunk. The first item is the business of the executive and technical experts. We may here discuss item two, and the following measures are suggested:—

1. *Instruction of Officers as to Procedure.*—The instruction of officers by lecture and demonstration of the properties and dangers from carbon dioxide, oxygen, and chlorine gases, and compressed-air illness. By this means a varying situation in the submarine would be appreciated and the correct orders issued.

The crew of a submarine are not only dependent on escape, but also on whether there is help at the surface. A premature exit would result in death from exposure, if not found, while a delayed escape incurs gas poisoning. To quote from a witness: "At 7 a.m. (sixteen hours after submergence) the situation had deteriorated and it was necessary to take deep and distressing breaths even when quiescent. Men were retching and yawning and watering from the eyes. One felt a great lassitude and it required a distinct effort to co-ordinate the mind and matter." Thus exit from a submarine should be timed before co-ordination between mind and muscle is lacking. To preserve his life a man must be able to think clearly, and it seems reasonable to suppose that tables could be drawn up, showing times of escape, which would serve for guidance to the Commanding Officer. The tables would provide data of cubic feet capacity of the vessel; number of men carried; and consequent increase in percentage of carbon dioxide hour by hour, which can be readily determined, for example:

1 man adds 1 cubic foot of carbon dioxide per hour
50 men add 50 cubic feet of carbon dioxide per hour

The submarine has a cubic foot capacity of about 25,000 cubic feet. Therefore at the end of one hour there would be 200 parts per 100,000, or 0·2 per cent, and 0·2 per cent of carbon dioxide at atmospheric pressure would equal 0·8 per cent at 4 atmospheres, or 99 ft.

2. *Simplification of Apparatus.*—In view of the mental effort required to co-ordinate the mind and limb, coupled with shock, it is necessary that the wearer of the apparatus should be able to manipulate the valves and cocks automatically without entailing a process of thought. Thus thorough training is requisite, frequent putting on of the apparatus, and periodic practice under water, in the D.S.E.A. which is at disposal at Depot.

By simplification of the apparatus mishaps due to forgetfulness would be avoided. Could the facepiece be made in one without separate nose-clips and mouthpiece stopcocks; and could valves be fitted so as to regulate the buoyancy?

3. *Breathing Mixture.*—As regards the gas breathed from the bag, it now appears that if pure oxygen is breathed by a man who is suffering from partial carbon dioxide poisoning he will suffer an onset of vomiting and prostration which will render him incapable.

Can the nature of the gas be improved upon by the substitution of a mixture instead of pure oxygen?

Mox Mohl, of Milwaukee, has invented a self-contained apparatus consisting of steel cylinders carried on the back, containing components of his own atmosphere.

By means of a distension mixture in one cylinder the wearer admits helium into the breathing bag to equalize the air pressure, then he supplies himself with oxygen from the other cylinder, in which mixture the partial pressure of oxygen approximates that of the normal atmosphere.

A similar mixture is 80 per cent helium and 20 per cent oxygen, in which decompression is hastened by 75 per cent. The mixture does not cause any physiological change, while nitrogen under pressure causes mental aberration and faintness. Moreover, whereas fat dissolves 5 parts of nitrogen, it dissolves only 1·7 parts of helium.

4. *A Submarine Decompression Chamber* (*Fig.* 567).—A submarine decompression chamber may be utilized for salvage operations if conditions are favourable; for example, if the submarine is lying on an even keel.

The chamber consists of a steel cylinder weighing 2 tons. It can be lowered over the side of the salvage ship by a derrick. There is a large door at the bottom through which a man can enter, and a smaller door at the top. Both doors are tested to 100 lb. sq. in. internal pressure. Air is supplied through an air-pipe with an inlet valve. Telephone and lighting are fitted. An oxygen breathing apparatus is carried. The chamber, with an attendant inside, is lowered into the sea with the lower door open. The supply and pressure of air is so adjusted as practically to keep the water out, but it may rise 1–2 ft. At the required depth the water is forced down until the air escapes through the lower door, when the supply can be readjusted. A ladder is put out through the lower door and the men from the submarine leave by equalizing the pressure on the escape hatch, and climb into the decompression chamber, which is then raised to the sea surface. The men may breathe oxygen from an apparatus to hasten decompression, which is about one-third of the time required when breathing air. The decompression according to the time tables can be continued subsequent to the lifting of the chamber into the ship at the surface, it being a sealed compartment. At the end of the time the men leave by the upper door, when the chamber can be used for the next batch.

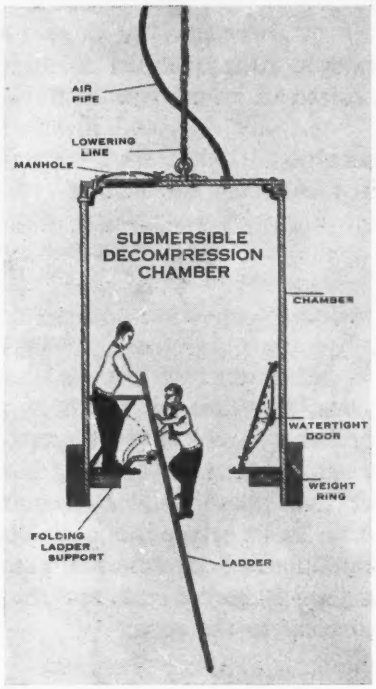

FIG. 567.—The submersible decompression chamber.

5. *The Diving Bell* (*Fig.* 568).—The diving bell, which has an open bottom, is lowered to the required depth at hydrostatic pressure and a man in the bell will breathe at the same pressure. Owing to the compressibility of air, water tends to rise in the bell as it is lowered. The rise is not uniform, but in accordance with Boyle's law. The diving bell has an open supply from the surface through the inlet valves. The diving bell is lowered to the submarine hatch, which is opened, and the survivors climb into the bell. The submarine has to be lying on an even keel and the bell kept level. Survivors would breathe oxygen from the apparatus in the bell if considered advisable, when being raised to the surface, to shorten decompression times.

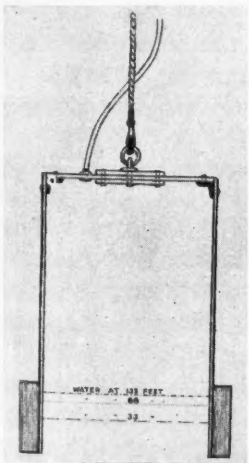

FIG. 568.—The diving bell.

6. *Absorbent for Purification and Dehumidification of Air.*—Soda lime apparatus for the absorption of carbon dioxide and dehumidification of air is fitted in submarines of the Italian Navy (Gimlette). Dehumidifiers are fitted in the more modern British submarines.

It has been shown that with the Admiralty policy the apparatus is not essential, and that fighting efficiency of the ship's personnel must take first place. Of course there will always be a school who will maintain

that by removal of carbon dioxide and water from the air in a submarine better fighting efficiency will be obtained. It is the tired and fatigued marksman whose torpedoes miss their objective.

7. *Diver as Part Complement.*—It is recommended that a submarine should carry a man qualified as diver in order that his experience at pressures may be utilized in an emergency.

8. *Shot Rope.*—A diver when returning to the surface from depths of more than 30 ft. delays his ascent on the 'shot rope' according to an authorized table in the *Diving Manual.*

Rising from 120 ft. after having been under pressure for 30 minutes, he
At 40 feet from the surface, stops for 1 minute
At 30 feet from the surface, stops for 2 minutes
At 20 feet from the surface, stops for 4 minutes
At 10 feet from the surface, stops for 4 minutes
Total time for ascent is 13 minutes.

If a survivor from a submarine could fully or partially conform with these stops, the time of which is variable, with the time under pressure, he would probably not incur compressed-air illness. Breathing the oxygen from his set would shorten the decompression. A shot rope from the surface to the submarine or vice versa would be required. The depths and time under pressure would have to be gauged, and the man would have to be in fair mental and physical condition. A diver would assist a man on a shot rope. It is probable, however, if the water were cold, that the man would be unable, owing to numbness of hands, to cling to the rope.

CONCLUSION

The escape apparatus is excellent in theory. Practically in several disasters only a few men escaped.

But it seems that a method of escape from a submarine will be so perfected that most of the inmates, in the event of mishap, will survive. Also, with the improvement in the class of vessel mishaps will become few and far between. In war there will, of course, be losses.

The problems are immense; the human being, with the most delicately adjusted respiratory centre, abhors a rapid change of environment, whether in respect of gaseous concentrations in the air, the blood, or in pressures and alveolar content of carbon dioxide, oxygen, and nitrogen.

It would appear that the escape compartment offers a more reliable means of escape than the escape chamber.

Acknowledgements.—The author desires to thank Surgeon Rear-Admiral H. E. R. Stephens, R.N., at whose invitation the article was written; and the Librarians of the British Medical Association and the Royal College of Surgeons of Edinburgh.

He also wishes to acknowledge the help he has received from the following sources: *Deep Diving and Submarine Operations*, by Sir R. Davis, published by St. Catherine's Press; Behnke, et al., *Amer. Jour. Physiol.*, 1934, **107**, 13; *Manual of Treatment of Gas Casualties*, 1930; *Manual of Diving*, B.R. 155/36; *Starling's Principles of Human Physiology* (Lovatt Evans), 7th ed., xxxvii; Surgeon-Commander Gimlette, R.N., *Jour. Roy. Naval Med. Service*, 1939, Jan.; Lecture Notes, Captain Damant, R.N. (Retd.).

DEEP DIVING

By Surgeon Lieutenant-Commander P. K. FRASER, M.D., Royal Navy

It is impossible in an article like this to confine oneself solely to the medical aspects of diving without considering the physics and physiology on which the subject is based. Similarly a technical description of the apparatus used may seem out of place; but on the integrity of the diving suit, air pump, valves, etc., depends not merely the diver's health, but his very life. The importance of prophylaxis in industrial medicine is being increasingly recognized, and this has been brought to a high pitch of perfection in the science of diving. How successful the various measures taken have been may be seen from the following records of recent diving operations.

In May of 1939, 71 dives up to a depth of 300 ft. were carried out by a group of 9 naval divers with only a few trivial mishaps, none of which required hospitalization or suspension from diving. The 'injector' type of diving suit was used with the Davis submersible decompression chamber. Subsequently, 92 dives from 150 to 180 ft. were made on the ill-fated *Thetis* with the standard dress. There was 1 unfortunate fatality which could not be attributed to the method used, and 2 minor cases of compressed-air illness.

THE EVOLUTION OF THE MODERN DIVING DRESS

Under-water operations doubtless began in prehistoric times with the recovery of personal belongings from shallow pools. This form of unassisted or natural diving still persists, and, despite increasing mechanization, it can be seen in the pearl and sponge industries to-day. Divers are also mentioned frequently in classical literature. About 300 B.C. the Rhodians formed what appears to have been a diver's guild, with a regular scale of fees for salvage work, varying according to the depth. This guild even quoted for work as little as 36 in. below the surface, which shows that the scope of salvage at that time was somewhat limited. Natural diving also played a prominent part in ancient naval warfare, and Herodotus mentions a diver who threw the ships of Xerxes into confusion by cutting their cables. As instancing the variety of early diving operations, Sir Robert H. Davis in his text-book of deep diving quotes Plutarch, who describes a fishing contest between Antony and Cleopatra. A diver was engaged by Antony to keep his hook supplied with fresh fish, but the deception was discovered and next morning a diver from the other camp was first on the scene with a salted and dried fish.

The earliest recorded account of supplying the diver with air is that of Aristotle, who mentions a method in which inverted jugs or buckets figure prominently. Pliny also mentions a form of breathing pipe which the diver held in his mouth. At a later age Leonardo da Vinci devised, among his many inventions, several types of submarine apparatus varying from a simple tube to an enclosed dress. Thereafter, many weird and wonderful contraptions were described in philosophical treatises, and astonishing performances were postulated by the originators.

Fortunately for these gentlemen and their dependants the experimental approach was not thought of, their inventions merely furnishing material for academic discussion.

From earlier efforts it gradually became obvious that to dive to any depth a continuous air supply under pressure had to be maintained, and subsequent suits were designed to enclose the head completely. The year 1819 saw the birth of the modern dress with the introduction of a suit and helmet by Augustus Siebe. With this model the diver was unprotected from the waist down, exhaust air escaping from under the jacket. Eighteen years later this was modified to enclose the diver, and, with minor improvements, it is now standard throughout the world.

EFFECTS OF PRESSURE UNDER WATER

At sea level there is a uniform pressure of 14·7 lb. to the square inch—1 atmosphere—due to the weight of the earth's gaseous envelope. In normal circumstances this ' absolute ' pressure is not appreciated as such, as the body is adapted to it, and pressure is usually taken to mean ' positive ' pressure, which is regarded as zero at sea level. To appreciate absolute pressure we can theoretically convert the thorax into a vacuum, when there would be an unsupported weight of about 2 tons on the ribs and thoracic muscles. In the case of a man under water there is the weight of the column of water above him to be considered. This must be counterbalanced by an equivalent air pressure in his suit, otherwise respiratory movements would be hampered and, in an extreme case, death by crushing would follow.

In salt water there is an increase in pressure of 0·44 lb. for every foot of depth, so that at 33 ft. there is a positive pressure of 14·7 lb. to the square inch, at 150 ft. 66·7 lb. to the sq. in., at 300 ft. 133·3 lb., and so on. At 150 ft., to overcome external pressure, there must be a pressure of 66·7 lb. to the square inch over the diver's chest and of course inside his lungs, and this is supplied from the surface.

It will be seen that the air pumps are working against a pressure corresponding to the depth of the diver. Consequently for deep work two or more pumps in series are necessary. In prolonged operations it is usual to store air in cylinders by means of a motor compressor and to run it off through a reducing valve as required. The air-pressure gauge is a useful method of watching the diver's depth.

PRESSURE AND GAS VOLUME

If a pressure of 14·7 lb. to the square inch, corresponding to a depth of 33 ft., be exerted on a given volume of gas the gas will be compressed to half its original volume. If the pressure be now doubled by increasing the depth to 66 ft. the volume of gas will be one-third, and at 99 ft. one-quarter. In other words, the volume of the gas varies inversely as the pressure and the density varies directly as the pressure. This is the well-known Boyle's law, the importance of which will be demonstrated.

When a diver descends to 33 ft. the volume of air in his suit is theoretically halved. Actually it is compensated by the air supply from the diving ship. If, however, the descent is from 169 ft. to 202 ft. the volume is only diminished by one-seventh. This explains the danger of a fall in shallow water. Should the diver accidentally drop from the surface to 33 ft. the diminution in volume inside

the suit will outpace the air supply from the surface and a ' squeeze ' will result. This will range from a sensation of weight on the chest in a mild case to death by crushing into the rigid helmet. Fatal cases have been reported where the diver has had to be dug out of his helmet.

The same thing happens in reverse order should the diver ' blow up ' out of control through closing his outlet valve. At first the ascent is gradual, but as the volume of air increases the suit becomes rigid and the diver is rushed to the surface in a crucified position. In a deep ' blow up ' he may smash his helmet against the bottom of the diving ship. It is extremely important after a ' blow up ' to gather in the slack of the air line and breast rope, otherwise the diver will fall to the bottom again and be squeezed. There is no danger of the suit bursting when over-inflated as air can escape from under the cuffs.

Pain in the ears due to alterations in pressure may sometimes be experienced during the first part of a rapid descent. It should be taken as a warning to retard the speed of the dive and inflate the Eustachian tubes. Failure to do this will lead to damage varying from a slight hæmorrhage down the handle of the malleus to rupture of the tympanic membrane. Most experienced divers clear their ears by swallowing, but some prefer to flatten the nose against the helmet and blow. Overt infection of the nose or throat should disqualify a man from diving.

THE MODERN DRESS

The standard pattern suit of twill and rubber as supplied to the Admiralty by Messrs. Siebe, Gorman & Co., of London, encloses the diver completely except for the head and hands. Rubber cuffs fit tightly at the wrists and there is a rubber ring at the neck which forms a watertight junction with the corselet or breast-plate. The rigid helmet engages with an interrupted thread on the corselet, while a drop-pin at the back prevents accidental unscrewing. Gloves can be worn in cold weather, but they are unpopular as they deaden the sensation of touch.

Air is supplied through a non-return inlet valve in the helmet and this guards against a sudden drop in pressure in the event of damage to the air line or reservoir. An outlet valve behind the diver's right ear maintains a pressure in the suit about half a pound above outside water pressure. This slight loading keeps the air well down over the chest and forms an elastic cushion against which to breathe. Incidentally, water pressure keeps the suit closely applied to the arms, legs, and abdomen. Should the dress be damaged, water is prevented from rising higher than the chest by pressure inside the suit, and at most the diver will only get a good soaking. Another standard Admiralty fitting on the helmet is a watertight telephone connexion. (*Figs.* 569, 570.)

There is a call button inside the helmet manipulated by the chin, and reception is through headphones. *Fig.* 571 shows the arrangement of the telephone fittings with a microphone on the right of the main window. It will be seen that incoming air is led down over the windows to prevent steaming. Another method of clearing the windows is to suck in sea water through the spit cock and squirt it on the glass.

To hold the diver down against a natural tendency to rise, special boots and weights are necessary. The boots with their leaden soles weigh about 18 lb. each, while the breast and back weights together amount to about 80 lb. It must not be forgotten that much of this weight is lost on submersion.

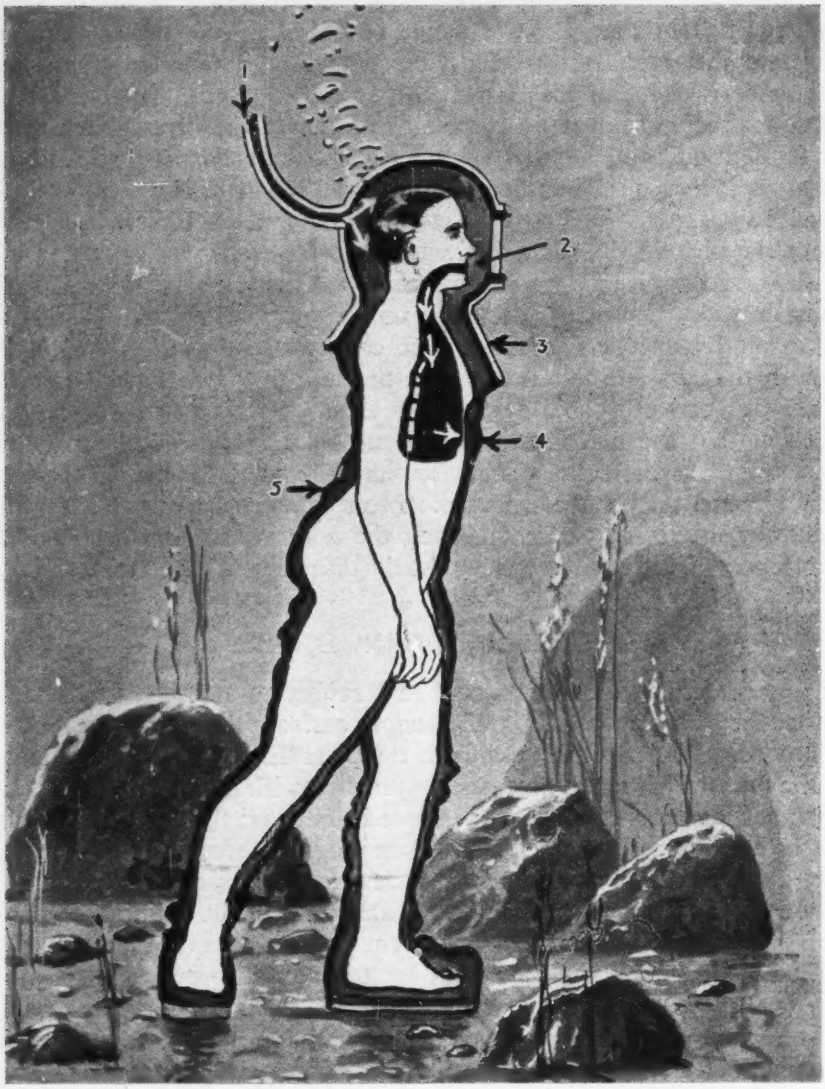

FIG. 569.—Diagrammatic section of diver under water. 1, Air coming from the pump must be at a pressure equal to that of the water round the diver, so as to inflate the dress and make breathing possible; 2, Pressure of air in the lungs is the same as the pressure of air in the helmet; 3, Rigid helmet and corselet; 4, Pressure of water (through dress) on outside of chest is balanced by equivalent air pressure inside; 5, Flexible dress.

The air pipe is made up of alternate sinking and floating lengths with a floating length nearest the diver to take the line out of his way. Similarly, a sinking length is connected to the air-supply to take the pipe down over the side of the diving vessel. Being a vital part of the equipment the airpipe should withstand a pressure of 200 lb. per sq. in. without bulging. Its strength lies in the ten layers of canvas, rubber, and steel wire which go into its construction. The breast rope which contains the telephone connexions can take a longitudinal strain of 1300 lb. without breaking.

Lastly to be considered among the standard articles of equipment is the shot rope, which runs from the surface to a weight on the bottom. This is a convenient form of submarine ladder and in dark water it acts as a ladder when exploring the sea bed. A steel wire under tension should never be used for this purpose as the bow-string effect may cause intestinal damage.

It is not usual to exceed 150 ft. with the suit described above, although it has been used at 300 ft., with a record dive of 340 ft., held by the British Navy. For dives over 150 ft. a special injector system of air supply is used whereby the air in the helmet is purified by passage over a canister of CO_2 absorbent. The effects of CO_2 under pressure will be discussed later.

FIG. 570.—Standard Admiralty pattern dress. The cuffs are turned back at the wrists and the air line and breast rope are led from the back of the helmet under the arms.

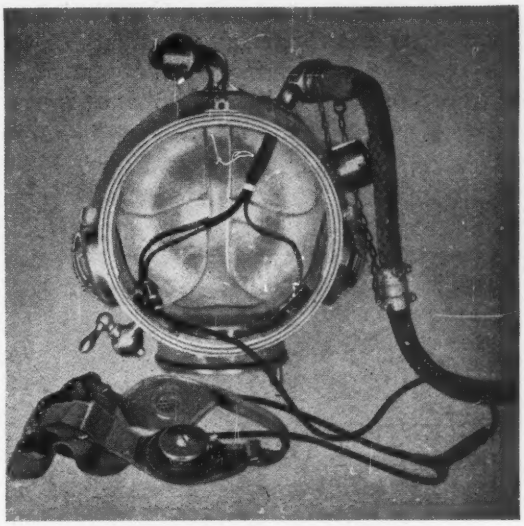

FIG. 571.—Interior of standard helmet.

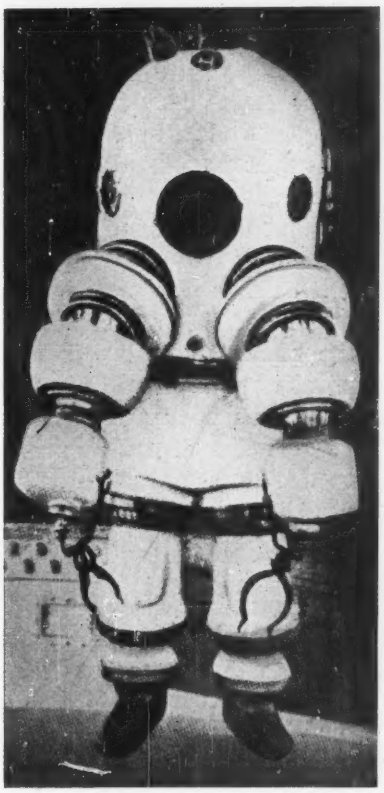

FIG. 572.—Armoured suit (Neufeldt and Kuhnke model).

ARMOURED DIVING DRESSES

To exceed 300 ft., which may be taken as the limit of the flexible suit, some type of armoured dress is necessary. At first glance this seems ideal for all types of work as the diver is kept at atmospheric pressure and air is supplied from cylinders carried in the suit. There is no danger of a squeeze and decompression is, of course, unnecessary. However, the object of diving is to perform work under water, and for this, flexibility and a free use of the hands are essential. With armour this flexibility is lost, as the diver is dependent on his lowering wire for stability and he cannot bend or thread his way through an opening. Only very simple operations can be carried out with pincers controlled from inside the suit. In a tideway the bulky arms act as fins, turning the diver in the direction of flow and away from his work. (*Fig.* 572.)

The Sorima Salvage Company, working on the wreck of the *Egypt*, attained a depth of 400 ft. with armour, but they abandoned it in favour of an observation chamber. These chambers are simple steel cylinders with circular windows through which salvage operations can be directed. The observer is kept at atmospheric pressure and there is a self-contained breathing system. A record descent of 3028 ft. has been made by Dr. Beebe, using a spherical chamber.

COMPRESSED-AIR ILLNESS

(*Caisson Disease*)

As improvements in apparatus increased the range of diving operations, so a strange set of symptoms appeared. On coming up from a depth agonizing pains were frequently experienced, and in severe cases paralysis and even death resulted. The descriptive term 'bends' was applied to the pains, and the paralysis was naturally named 'diver's palsy'. These conditions were ascribed to the effects of pressure, but the underlying cause was first suspected by De Mericourt (1860), who likened the diver under pressure to a bottle of soda water. Later, Paul Bert (1878) worked out the mechanism of this new entity and laid the foundation for all future studies.

Respiration under pressure will lead to solution of nitrogen in the blood and tissues. Oxygen and CO_2 are also dissolved, but except at very high and prolonged pressures the body can deal with them and they do not cause symptoms. A sudden removal of pressure will liberate dissolved nitrogen in the form of bubbles, and these have been observed experimentally in the capillaries of the frog's web. The degree of solution depends on the pressure and the length of exposure, while the rate depends on the vascularity of the part. Thus some parts will be fully saturated in a short time while others will take as long as five hours. Conversely the rate of desaturation is very variable.

To combat compressed-air illness Paul Bert introduced a slow uniform rate of decompression and this was a most important prophylactic advance. He also advocated the administration of oxygen as a means of washing out dissolved nitrogen. Despite these measures, however, cases still continued to appear, and in 1906 a committee of investigation was set up by the Admiralty. Professor J. S. Haldane, the physiological member of this committee, introduced the 'stage' method of decompression whereby the diver comes up in steps, and this has been universally adopted.

The reasoning behind this system is interesting. It is known that the blood can hold nitrogen in solution at a pressure equal to 2 atmospheres absolute without the formation of bubbles. In other words, dives up to 33 ft., no matter how prolonged, require no decompression. Applying this to greater depths, Haldane argued that it would be safe to halve the absolute pressure, allowing the diver a certain time at each stage to get rid of excess nitrogen. Thus a man can be btought directly from 6 atmospheres (165 ft.) to 3 atmospheres (66 ft.), and so on, with perfect safety. This 2 : 1 ratio holds good except at very great depths, when it becomes 1·75 : 1. The actual tables were worked out by experiments on men and animals, and the value of exercise in hastening decompression was demonstrated.

Various investigations have been carried out to eliminate the nitrogen hazard, and American workers report encouraging results from helium-oxygen mixtures. Helium is much less soluble in the blood than nitrogen, and being inert it has no effect on the body. The decompression period required is very much shortened. It is not a workable proposition in this country, however, on account of the difficulties of supply.

PREVENTION OF COMPRESSED-AIR ILLNESS

Oxygen in Decompression.—It was pointed out by the Admiralty committee in 1907 that the administration of pure oxygen would half the time of the decompression stages in which it was used. There was, however, the known danger of oxygen poisoning under pressure. This was fully worked out by another committee in 1930, and it was found that it was not safe to breathe pure oxygen at a pressure greater than that corresponding to a depth of 60 ft. *Table I* shows the decompression table for a depth of 200 ft., using oxygen. The old method of

Table I.—DIVERS' DECOMPRESSION TABLES FOR 200 ft.

AIR					OXYGEN								
Time on Bottom, including Descent	Ascent	90 ft.	80 ft.	70 ft.	60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	Total Air	Total Oxygen	Aggregate Time
min.	min.	min.	min.	min.	min.	min.	min.	min.	min.	min.	min.	min.	min.
12	3	–	–	1	1	1	2	3	4	5	4	16	20
16	3	–	–	1	1	2	3	3	6	6	4	21	25
20	3	–	–	1	2	2	3	3	7	8	4	25	29
25	3	–	–	2	2	3	4	5	8	10	5	32	37
30	2	–	1	2	4	4	6	7	11	12	5	44	49
35	2	–	1	3	5	6	7	8	14	15	6	55	61
40	2	–	2	3	5	6	8	9	15	17	7	60	67
45	2	–	2	3	6	7	9	11	16	20	7	69	76
50	2	–	2	4	7	8	9	13	17	22	8	76	84
55	2	–	2	5	7	8	10	15	20	26	9	86	95
60	2	–	3	5	7	8	11	16	24	29	10	95	105
80	2	–	5	6	9	11	15	18	34	34	13	121	134
100	2	–	6	6	11	14	16	25	34	34	14	134	148
120	2	3	5	8	13	15	21	29	35	35	18	148	166
180	2	3	9	12	18	24	28	30	35	35	26	170	196

decompressing the diver on his shot rope is somewhat exhausting, especially in cold and tidal waters.

Submersible Decompression Chamber.—The use of oxygen in decompression necessitates some form of underwater chamber, and the type in common

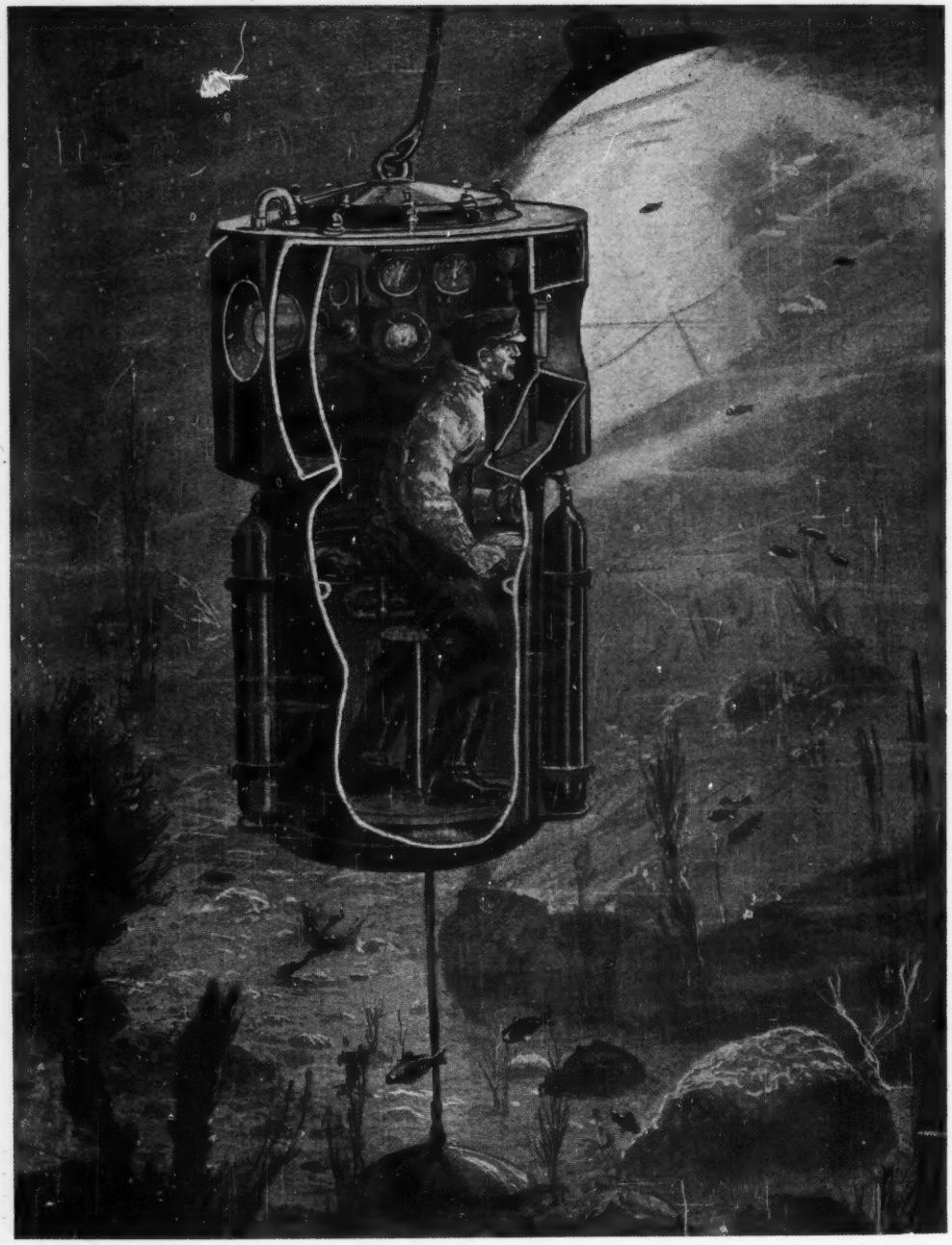

FIG. 573.—Observation chamber (Siebe, Gorman & Co.).

use was devised by Sir Robert H. Davis, of Siebe, Gorman & Co. It is rather like a diving bell, having an air-supply from the surface, telephone installations, a lighting system, clocks, pressure gauges, etc. The chamber is lowered to 60 ft.

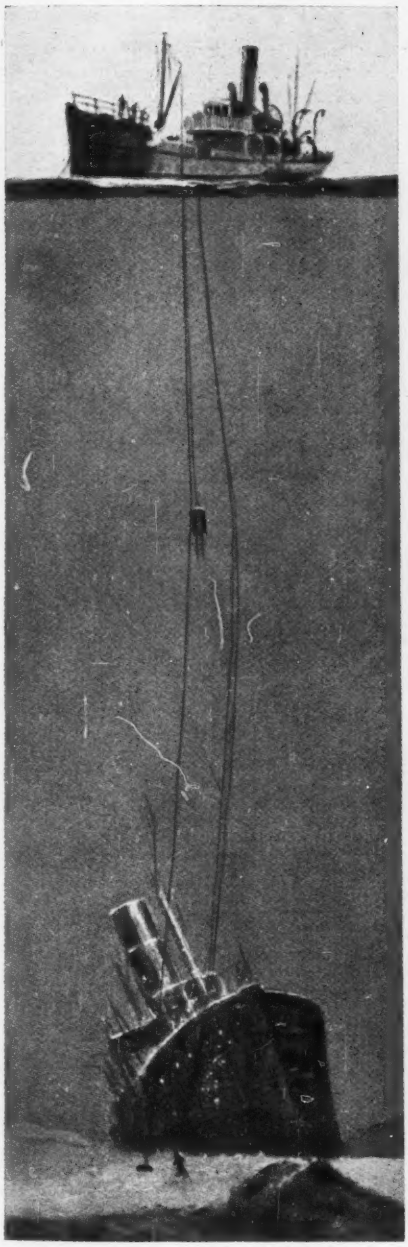

FIG. 574.—Davis submersible decompression chamber lowered ready for use.

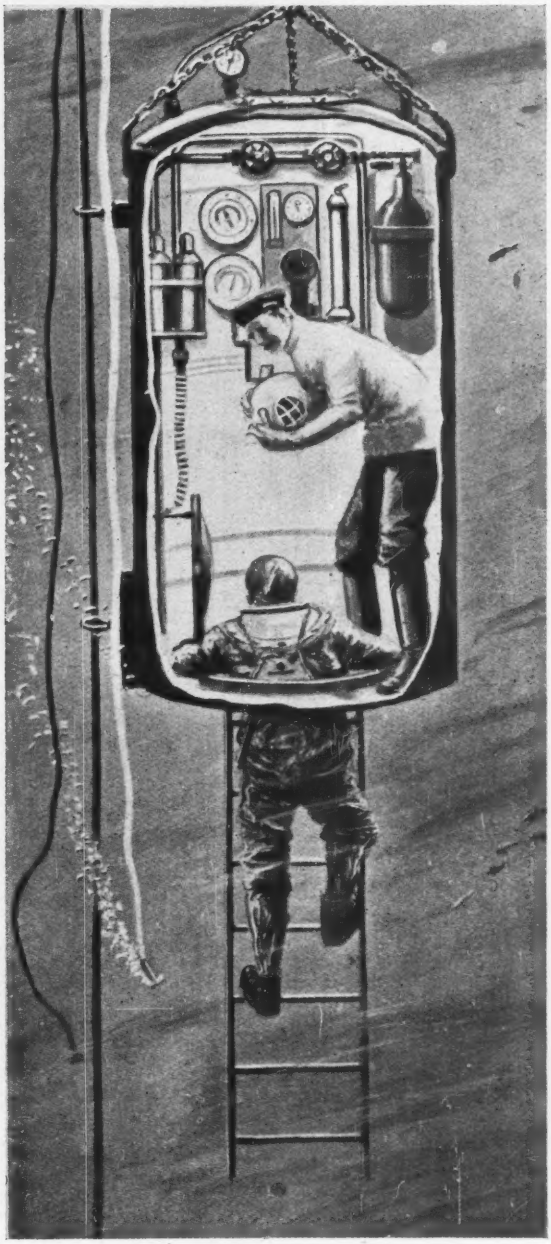

FIG. 575.—Diver entering chamber.

and cleared of water by air pressure. When the diver is safely inside, the lower door is shut and the whole is hoisted on board the diving ship, where decompression

is carried out according to scale by the attendant. An idea of the working of this system will be gained from the accompanying illustrations (*Figs.* 573–576).

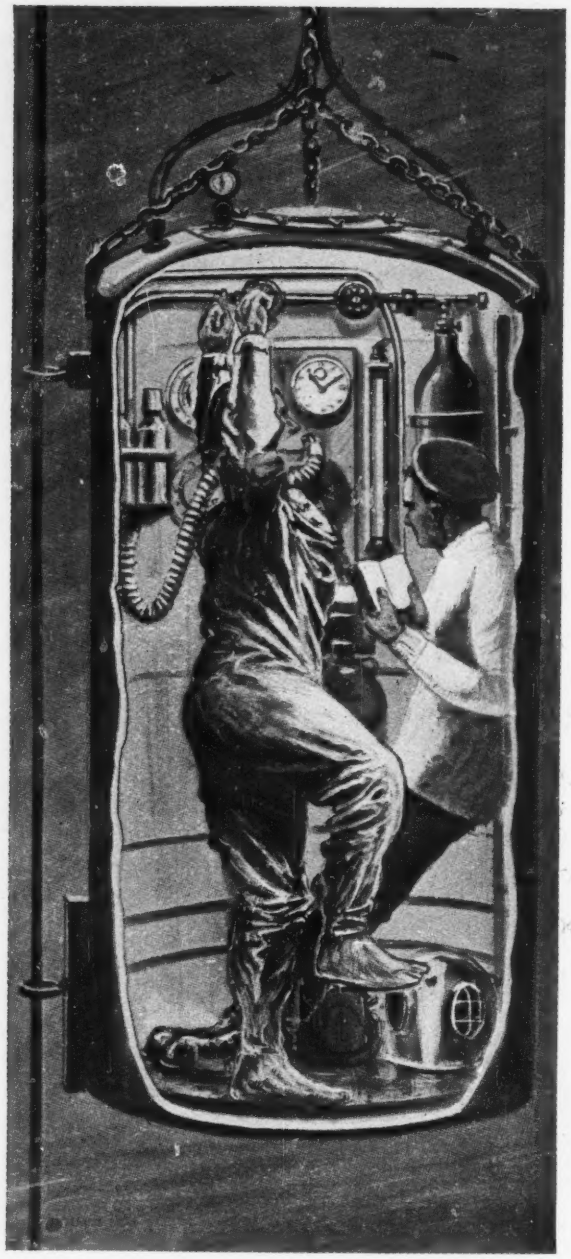

FIG. 576.—Diver breathing oxygen in chamber.

The diver breathes oxygen by means of the 'Novus' apparatus (*Fig.* 577). For the first five minutes he inhales from the bag and breathes out through the by-pass valve. Thereafter he exhales over a canister of CO_2 absorbent. The bag

is emptied at each stage in decompression to get rid of exhaled nitrogen and refilled with pure oxygen. (*Fig.* 578.)

Table II.—DECOMPRESSION TABLE FOR COMPRESSED-AIR ILLNESS

Pressure in the Chamber	*Maximum Rate of Fall in Pressure*
Between 100 lb. and 90 lb.	4 lb. in 1 minute
Between 90 lb. and 75 lb.	2 lb. in 1 minute
Between 75 lb. and 60 lb.	1 lb. in 1 minute
Between 60 lb. and 45 lb.	1 lb. in 1½ minutes
Between 45 lb. and 30 lb.	1 lb. in 3 minutes
Between 30 lb. and 15 lb.	1 lb. in 5 minutes
Between 15 lb. and zero	1 lb. in 8 minutes

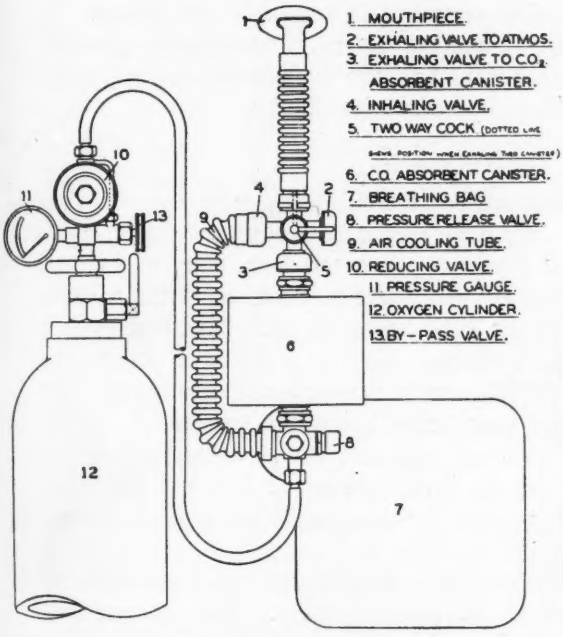
1. MOUTHPIECE.
2. EXHALING VALVE TO ATMOS.
3. EXHALING VALVE TO CO_2 ABSORBENT CANISTER.
4. INHALING VALVE.
5. TWO WAY COCK (DOTTED LINE SHOWS POSITION WHEN EXHALING THRO CANISTER)
6. CO_2 ABSORBENT CANISTER.
7. BREATHING BAG
8. PRESSURE RELEASE VALVE.
9. AIR COOLING TUBE.
10. REDUCING VALVE.
11. PRESSURE GAUGE.
12. OXYGEN CYLINDER.
13. BY-PASS VALVE.

FIG. 577.—Novus oxygen breathing apparatus.

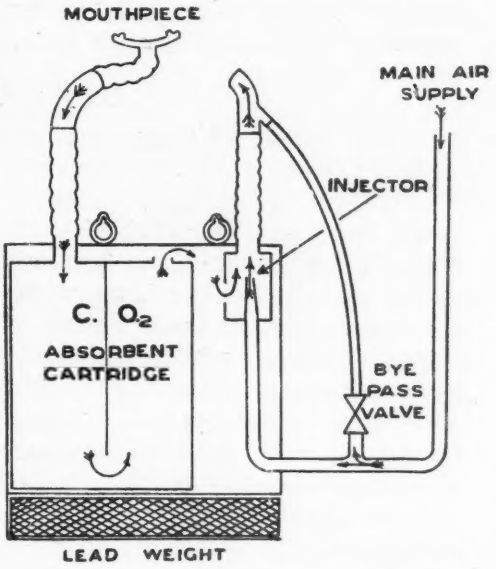

FIG. 578.—System for purifying the diver's air.

The advantages of the submersible chamber are manifold. In cold water the diver is spared a long wait on the shot rope and he can get a warm drink on entering the chamber. During intensive operations he can be left to decompress out of the way of further divers. Should a gale spring up the diving ship can run for shelter with the chamber safely on board.

During operations on the *Thetis* it was found inadvisable to use the chamber continuously on account of the strength of the tides and the conglomeration of salvage craft, and another method was introduced. The diver was brought straight to the surface and recompressed on board in a special chamber to a depth corresponding to his first stop. Decompression then proceeded in the normal way, using the Novus apparatus. It may be mentioned that the nose is closed by a spring clip during oxygen inhalations.

TREATMENT OF COMPRESSED-AIR ILLNESS

The infallible treatment for this condition is *recompression.* This can be done by sending the diver down again or by putting him in a special recompression

chamber. The pressure should be raised till all symptoms disappear, when decompression should be carried out according to a special table. If symptoms reappear the pressure will have to be raised again and the decompression time increased by 50 per cent. A bad case of ' bends ' will take five or six hours to cure. Massage of the affected part will aid resolution, and heat in the form of a moist compress or hot-air cage is comforting. Paralysis of both legs should be taken as an indication of nitrogen bubbles in the spinal cord.

It cannot be too strongly stressed that speed in recompression is the main essential in the treatment of compressed-air illness. The longer a man is kept at atmospheric pressure the more difficult will he be to treat. In the case of an unconscious diver when no recompression chamber is available, he should be lowered gently to the bottom in the care of another diver. Theoretically ' bends ' can affect any part of the body, but they are commonest in the ligamentous insertions round joints. Undue exertion and cold are predisposing factors.

OXYGEN POISONING

The toxicity of oxygen was demonstrated by Paul Bert, who showed that animals exposed to high pressures of the gas developed convulsions and finally died. Working on this problem, Bornstein, of Hamburg, and von Schrotter, of Vienna, found that 3 atmospheres absolute of pure oxygen could be breathed without harm for at least 30 minutes, and Bornstein at the Elbe tunnel in 1909 breathed oxygen at this pressure for 45 minutes without ill effects. Now at a depth of 300 ft. the inspired air contains the equivalent of slightly over 2 atmospheres absolute of pure oxygen; consequently, in prolonged and very deep diving the danger of oxygen poisoning arises. Pure oxygen under pressure may also act as a lung irritant if breathed for a long period. Working on the U.S. submarine F.4, off Honolulu in 1915, one of the divers became fouled at 250 ft. for three hours and later developed double pneumonia.

The 1930 Admiralty committee, after extensive experiments, set 3 atmospheres absolute of pure oxygen for 30 minutes as the limit of safety. This corresponds roughly to the 60 ft. mentioned in connexion with oxygen in decompression. It will be noticed in the decompression tables that the stages from 60 ft. to 30 ft. are comparatively short.

CARBON-DIOXIDE POISONING

At sea level the percentage of CO_2 in the air is about 0·03 per cent, at which concentration it causes no effect. When this is increased to 3·0 per cent respirations become twice as deep as normally, with severe panting at 10 per cent. Loss of consciousness occurs at about 12 per cent. It will be seen that at 300 ft. (9 atmospheres positive approximately) a concentration of 0·03 per cent CO_2 in the inspired air has the same effect as breathing 0·27 per cent of the gas. In addition, there is the cumulative effect of the inspired air in the helmet.

Respiration under pressure increases the oxygen tension of the venous blood, consequently there may be a dangerous accumulation of CO_2 in the tissues. The slightest increase in the quantity of gas in the helmet may cause unpleasant symptoms. Experienced divers when they feel distressed will call for a ' flush through ' of air from the surface. This invariably removes the excess. The injector system, whereby the diver carries a canister of CO_2 absorbent on his back, is most satisfactory in deep work.

SELECTION AND SUPERVISION OF DIVERS

Only perfectly healthy men should be selected for diving instruction. They should be under 30 years of age and should be muscular without being unduly fat. As modern diving under experienced supervision has not the slightest tendency to shorten life, the age limit for work depends on the individual. In difficult operations experience counts for everything. Very short and very tall men are unsatisfactory, as the standard equipment does not fit them comfortably. A nervous disposition or a history of nervous instability should disqualify, as this is bound to crop up in prolonged work, especially in dark water. Lung expansion should be good and men of a sober and quiet disposition are preferred.

Divers should be examined frequently and they should be instructed in the medical aspects of their speciality. Such a knowledge inspires confidence and makes for co-operation. Young divers should be tested under pressure to weed out those with a predisposition to compressed-air illness. In continuous salvage work symptoms of exhaustion or staleness are dangerous, as they render the men accident prone. A rise in systolic blood-pressure should be taken as a danger signal. Comfortable and cheerful quarters are essential, and frequent periods of recreation and leave should be arranged.

SUPPLEMENTARY AIDS TO DIVING

With the Admiralty pattern suit the weight of the helmet is taken by a padded ring worn over the shoulders. Woollen drawers and jerseys are useful in winter and a binder of flannel or other material keeps the lumbar region warm. To prevent numbing of the hands in cold water, they can be rubbed with menthol paste or other stimulating ointment. During Arctic operations, dehydration and heating of the air are essential to prevent ice forming at the outlet valve.

When a diver inflates his suit to lighten himself the helmet may rise over his head; a jock strap attached back and front to the corselet prevents this. Short men are especially liable to this inconvenience. Another useful aid when working in an enclosed space is a weighted belt instead of the normal weights. This enables the diver to wriggle through an opening without danger of being jammed on the return trip. The knife, without which no diver is complete, is not used normally for fighting giant squids, but is essential when working round wrecks with trailing ropes.

COMMENTARY

This article is not intended as a complete review of the science of diving. For a wider survey the standard works should be consulted. We hope, however, to have demonstrated the difficulties which beset diving operations. To the misinformed such things as tides, compressed-air illness, squeezes, etc., do not exist, and this information may lead to a more rational outlook on salvage work.

Acknowledgements.—We are indebted to Sir Robert H. Davis, head of the firm of Siebe, Gorman & Co., Ltd., London, for permission to quote from his book, *Deep Diving and Submarine Operations*. The illustrations were kindly supplied from the same source. From time to time the Admiralty committees on deep diving have furnished reports, and these have been freely consulted in the compilation of this paper.

SHORT NOTES OF RARE OR OBSCURE CASES

SPONTANEOUS RUPTURE OF THE ŒSOPHAGUS

By P. C. MALLAM, H. A. B. WHITELOCKE,
AND A. H. T. ROBB-SMITH, Oxford

Since the condition was first described by Boerhaave in 1724, there have been only 40 cases published of spontaneous rupture of the œsophagus, and it is evident that a condition of such rarity should be accurately recorded whenever it occurs.

The original case was that of Baron Wassenaer, Grand Admiral of the Dutch Navy, who was a notable glutton, and it was his custom when he had consumed more than his stomach could comfortably carry to relieve himself in the Roman fashion by titillating his uvula. One day in 1723 he had enjoyed a particularly ample meal but recourse to his usual manœuvre evoked no response; accordingly, he took a gentle vomit and was immediately overwhelmed by an agonizing pain in the chest with a sensation of bursting; he collapsed and rolled about his room in exquisite agony, covered with sweat, retching, but not vomiting. Hermann Boerhaave was consulted but was unable to ease his pain, and after eighteen hours of torment the unhappy man died. The great physician, feeling that the condition was as obscure as it was disastrous, asked the relatives to allow a post-mortem examination. This was granted and it was found that the œsophagus had ruptured a short distance above the diaphragm and that gastric contents were free in both pleural cavities. Boerhaave felt that such a condition deserved recording and it forms the basis of the first of the only two post-mortem accounts which he published. It appeared in the form of a pamphlet entitled *Atrocis, nec descripti prius, Morbi Historia*, published in 1724.

CASE REPORT

Clinical History.—The patient was a man aged 44, an instructor in physical training. He was rather fat, but physically extremely well developed. The history of the illness was that he had taken an ordinary dinner and attended a boxing match as a spectator. On returning from this he had taken a rather heavy supper and retired to bed about 11.30 p.m.; he was temperate in his habits and had in no way exceeded during the evening. He woke up about 2.15 a.m. feeling perfectly well and drank a glass of water and immediately was struck down with desperate pain. He was seen by one of us (P. C. M.) at 2.40 a.m. and at that time he was extremely restless, moaning, and in great agony, unable even to give his name or age. On examination his abdomen was distended, hard, and rigid; his lungs appeared normal except for a few crepitations at the bases; and his tongue was clean.

The patient was moved to a Nursing Home and was seen at 4.15 a.m. in consultation with the surgeon (H. A. B. W.). An acute abdominal emergency, probably a perforated gastric ulcer, appeared the obvious diagnosis, though he was complaining of pain posteriorly in the lower thoracic region.

He was given ½ gr. morphia and operated on at 5.5 a.m., the usual epigastric incision being made for an upper abdominal exploration. A slight amount of free

fluid appeared when the peritoneum had been opened. All the upper abdominal viscera were carefully examined and the cause of the condition was still a mystery. The lower abdomen was next explored and some more clear fluid mopped out. It was decided to examine the appendix as a probable alternative diagnosis, but this also was natural. The lower incision was subsequently drained and the patient returned to bed within twenty minutes of induction of anæsthesia.

When he was seen at 9 a.m. he was still in great pain and could not bear to lie down though he had had three ½-gr. doses of morphia during the previous six and a half hours. His pulse was poor, he was in a clammy sweat, and his temperature had risen to 100° F., although originally it was normal.

At midday he was almost pulseless and it was obvious that he was dying. His pain persisted and his chest still showed no gross signs, although his breathing was poor. The exhibition of cardiazol and coramine failed to have any effect on the heart and he died just before 2 p.m., almost twelve hours after the onset of symptoms.

From the medical point of view the striking features about the case were :—

1. That the patient remained in acute agony the whole time, despite large doses of morphia.
2. That he could not lie down or keep still.
3. That he suffered from extreme thirst; this was possibly due to the morphia, though the symptom manifested itself before any morphia had been given.

POST-MORTEM EXAMINATION (R.I. 280/38).—The examination was carried out eighteen hours after death. The body was that of an obese man with recent midline and right inguinal incisions. There was an extreme degree of emphysema of the subcutaneous tissue of the face, neck, and chest, and although there was discoloration of the skin and evidence of proliferation of gas-forming organisms, yet the degree of emphysema was greater than could be explained by infection alone.

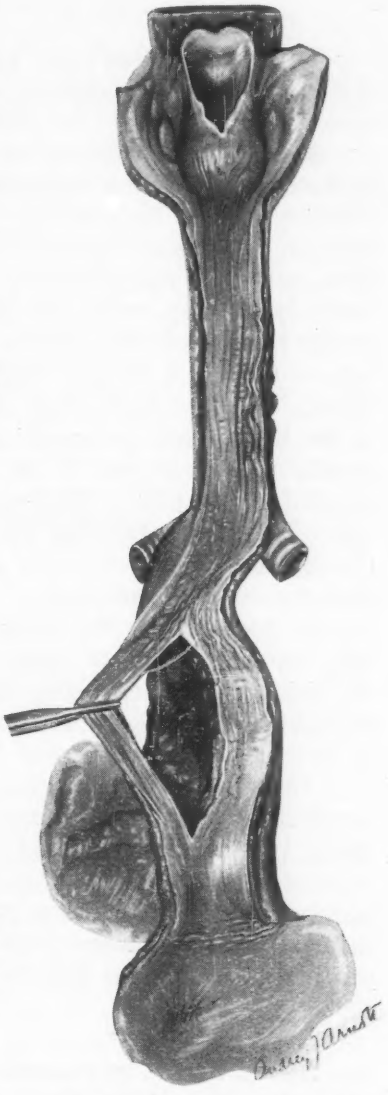

FIG. 579.—Drawing of the œsophagus, showing the longitudinal tear in the anterolateral wall.

In both pleural cavities there were effusions of brownish fluid smelling of gastric contents; this was more marked on the left than on the right. Examination of the œsophagus revealed a longitudinal linear tear through all the coats. It was 6 cm. long and lay to the left of the midline. The lower limit of the tear was 4 cm. above the cardia. There was no evidence of any other abnormality in the œsophagus, and undigested foodstuff was present in the œsophagus, in the tear, and in the posterior mediastinum, particularly on the left

side. The stomach was markedly distended and contained a partially digested meal of ample amount.

The other organs presented no abnormality.

Sections of the œsophagus showed no scarring or pathological process apart from a post-mortem infection by gas-forming bacilli.

DISCUSSION

The condition has been well reviewed by Girard (1934) and Terracol (1938), and it will only be necessary to emphasize the salient features, which are well exemplified in the case described.

Almost all the subjects have been obese men usually given to good living and in the majority of cases addicted to the bottle (in only 4 other cases has there been a history of total abstinence). The attack has commonly come on after an ample meal, in some cases associated with vomiting or eructation. In a few cases it has been associated with vomiting due to a cerebral lesion. The illness commences suddenly, with agonizing pain either in the epigastrium or substernal region; the patient is shocked, with pallor, sweating, a rapid small pulse, cold extremities, and intense thirst; a surgical emphysema develops first around the neck and then spreads over the chest, face, and limbs. Clinical examination reveals fine rales at the bases (most commonly the right) and signs of a hydropneumothorax. In certain cases aspiration of the chest has revealed the presence of an acrid fluid containing foodstuffs and having the characters of gastric juice. In only one case—that of Walker (1914)—has the diagnosis been made during life. All attempts at therapy have been unsuccessful and death has occurred in from 24 to 48 hours after the onset of symptoms.

At post-mortem the lesion is almost invariably a longitudinal tear 1–8 cm. long involving all three coats and usually occurring on the posterior surface to the right, immediately above the cardia. Histological examination of the tear has revealed in certain cases old inflammatory or fibrotic changes, but in many cases no pathological process could be demonstrated.

The aetiology is obscure; experimentally it has been shown that a weight between 0·5 and 1·5 kg. is necessary to produce rupture of a healthy œsophagus, and it is generally considered that this is much greater than occurs in vomiting; some authors have insisted that the œsophagus was previously diseased and this may well be the case, but it seems probable that if the circular muscles are in spasm and there is a sudden contraction of the diaphragm the strain would be such as to produce a lesion of the kind found in spontaneous rupture of the œsophagus.

SUMMARY

A fatal case of spontaneous rupture of the œsophagus which had the classical symptomatology is described, and a brief account of existing knowledge of the condition is presented.

We are indebted to Miss Audrey Arnott for the drawing of the specimen.

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REVIEWS AND NOTICES OF BOOKS

Guy's Hospital Reports. Edited by Sir ARTHUR HURST, D.M. Vol. LXXXIX (Vol. XIX, Fourth Series, No. 2, April, 1939). 9½ × 6 in. Pp. 121–249. Illustrated. 1939. London: Headley Brothers. Issued quarterly. Single numbers 10s. net; Annual subscription 30s. net.

THIS volume opens with a very fine appreciation of the late Sir Cooper Perry by Mr. H. L. Eason, and it contains several short studies of clinical cases as well as two longer and more important contributions, one from the Orthopædic Department of the Hospital, and the other dealing with the epidemiology of acute anterior poliomyelitis, more especially in regard to the management of the disease in schools, written by Dr. R. E. Smith, Medical Officer of Rugby School. Attention is drawn to the probability of an intestinal portal of entry for the infection, and the article contains much information which will be of value not only to school medical officers but also to those who have the responsibility of advising parents whose children are attending a school where the disease has appeared.

Mr. Lambrinudi and Mr. Stamm have put together a review of the work of the Orthopædic Department which is just the sort of article hospital reports ought to contain. It indicates clearly the changes which have taken place at Guy's and elsewhere during the past few years in the treatment of deformities of the feet, and of scoliosis, and how physiological principles underlying muscular action have been applied to the management of paralytic deformities, and of fractures, and also to manipulative surgery. This is a thought-provoking paper of which the value is greatly enhanced by a number of simple but very clear drawings.

Of the shorter articles the most interesting deals with subacute ulceration of the stomach associated with achlorhydria, a condition first described in the Reports of a sister hospital—Sir Arthur Hurst pays a graceful tribute to Rodgers and Avery Jones of St. Bartholomew's in awarding them priority in this matter. The remaining papers, by Drs. H. A. Hamilton and J. Whillis on a case of ovarian pregnancy, by Sir Arthur Hurst on regional colitis, and by Drs. P. M. F. Bishop and B. G. Shapiro on Cushing's syndrome, all contain points of considerable interest and importance.

War Wounds and Air Raid Casualties. Articles republished from *the British Medical Journal*. 8½ × 5½ in. Pp. 258 + x. Illustrations. 1939. London: H. K. Lewis & Co. 10s. 6d. net.

THE twenty-three chapters which comprise this book are written by eminent authorities. Many younger surgeons have felt that the experience of those of the senior generation who saw war service should be detailed for their instruction. The reviewer, however, has received the unhappy feeling that this experience could have been better presented. Some authors seem to have turned to their 1914–18 experience and resolutely shut their eyes to any subsequent knowledge. This is particularly seen in the chapter on Tetanus and Gas Gangrene. Contradictions occur; the reader can choose between skeletal traction and the Böhler-Braun splint advocated in War Surgery in Spain, and the Thomas splint and skin traction (the author persists in writing "extension") in Injuries of the Lower Limb. It is remarkable that there is no reference to drugs of the sulphanilamide-sulphapyridine group in the chapters Treatment of Wounds in War, Principles of Treatment in Gunshot Wounds of Joints—Injuries of the Lower Limb. The sole index reference to these drugs is, oddly enough, to the chapter on Tetanus and Gas Gangrene. Chapters on war-time anæsthesia and face and jaw injuries are not included. It is doubtful whether the practitioner will wish to read a section Is Gas Warfare Inhuman?, however interesting this may be in its ethical aspects. Apart from the criticisms above, the book is useful and it has received the blessing of the Director-General of the Army Medical Services.

A Text-book of X-ray Diagnosis. By British Authors. Edited by S. COCHRANE SHANKS, M.D., M.R.C.P., F.F.R., PETER KERLEY, M.D., M.R.C.P., F.F.R., D.M.R.E., and E. W. TWINING, M.R.C.S., M.R.C.P., F.F.R., D.M.R.E. In three volumes. $9\frac{3}{4} \times 7\frac{1}{4}$ in. Vol. III, Skeletal and Nervous Systems. Pp. 800 + xiv, with 710 illustrations. 1939. London: H. K. Lewis & Co. 63s. net.

THE third and final volume of this work has now appeared, and fully maintains the standard set by the previous two volumes. As the editors say in their preface, the production of this volume has been a lengthier process than in the case of the first two, largely because of the time involved in correlating the work of sixteen contributors. These not only include many of the leading radiologists in this country, who have been chosen for their special knowledge in particular branches of the subject, but also eminent clinicians, namely, Hugh Cairns, Donald Hunter, C. Jennings Marshall, and J. St. George Wilson, together with S. L. Baker, Professor of Pathology at the University of Manchester. From such a team of collaborators has resulted a comprehensive and harmonious survey of the radiological and clinical aspects of the subject-matter dealt with.

The volume is divided into six parts, dealing with the Central Nervous System; the Accessory Nasal Sinuses, Labyrinth, and Mastoid Process; the Bones and Joints, and Soft Tissues (including a short section on the Localization of Foreign Bodies); the Teeth and Jaws; the Eye; and Cineradiography. Some of these parts are divided into sections, and the volume contains some sixty chapters in all. At the end of each section and of some of the chapters there is a very full and most useful bibliography, and a good index completes the work.

Part I, on the Central Nervous System, by Hugh Cairns and M. H. Jupe, gives in 182 pages a very thorough account of all the latest work on the radiological investigation and features of cranial, intracranial, and spinal cord lesions, with sufficient details of the technique of examination and of the underlying pathology to render this section of supreme interest and value to all interested in this branch of medicine and surgery. The chapters are fully illustrated with excellent reproductions and radiographs, post-mortem specimens, and explanatory diagrams.

Part II on the Accessory Nasal Sinuses, etc., is ably contributed by Dr. Graham Hodgson, whose work on this region is already well known.

Part III is by far the largest portion of the volume; it consists of more than four hundred pages, and is the work of as many as thirteen contributors. Its arrangement into eleven sections is well thought out, and every variety of bone and joint condition, normal, congenital, traumatic, inflammatory, static and paralytic, constitutional and neoplastic is considered and well illustrated by experts in their respective subjects. One is particularly glad to see included in Section II an excellent description of the general pathology of bone by Professor S. L. Baker, without a knowledge of which a true understanding of the radiographic appearances cannot be fully appreciated. The subjects covered in this part are too many to be mentioned in detail, but Section VIII on Diseases of Bones and Joints of Constitutional or Unknown Origin, by Donald Hunter and M. H. Jupe, is a highly interesting and illuminating contribution to the study of a group of diseases some of which have until recent years been little understood.

The section on Tumours and Cysts of Bone is methodically and capably dealt with by Dr. R. E. Roberts, who, in the chapter on Malignant Tumours of Bone, wisely chooses the classification adopted by the American Bone Registry. He emphasizes the fact that many of the radiographic changes seen are often due more to the reaction of the bone to the invading tumour than to the tumour itself. On the general question of bone reaction to malignant disease, no full explanation appears yet to have been given by the pathologists or biochemists of the alternative types of appearance so well known to the radiologist, namely the so-called osteolytic and osteoblastic (or sclerosing) response. Why do secondary deposits from a malignant prostate, for example, usually produce a sclerosing type of response while occasionally the opposite effect is seen, or even a mixture of the two? To say that in one case the bone is stimulated to respond while in the other no response is elicited and the invaded bone is merely replaced by malignant tissue, is merely stating the problem in another way. There appears to be little or no evidence that a scirrhous carcinoma of the breast is more likely to produce a scirrhous reaction in bone. The time-factor may well be the deciding point in the process. This interesting problem is discussed by Professor Baker in Section XVII, Chapter XIX, but further information would be welcome.

The subject of the Teeth and Jaws in Part IV has been most carefully and thoroughly dealt with by H. M. Worth, and again one must draw attention to the quality of the illustrations, the technical excellence of which in the whole work reflects the greatest credit on the editors, the block-makers, and the printers.

Part V, on the Eye, deals mainly with the details of accurate localization of foreign bodies in this organ; and to complete this most extensive and up-to-date work Dr. Russell Reynolds in Part VI contributes an excellent description of the latest developments and applications of Cineradiography, a subject in which he has achieved international fame.

The general impression gained from a survey of the whole work is that the editors have produced a text-book which will be standard for many years, and which fully upholds the high traditions of British medicine.

The Clinical Diagnosis of Swellings. By C. E. CORRIGAN, B.A., M.D., F.R.C.S. (Eng.), Lecturer in Surgery, University of Manitoba; Assistant Surgeon and Director of the Out-patient Department, St. Boniface Hospital. 9⅛ × 6 in. Pp. 313 + viii, with 120 illustrations. 1939. London: Baillière, Tindall & Cox. 18s. net.

IT is encouraging to find a teacher of surgery who insists so strongly on the examination of the patient as to write a book of 300 pages entirely devoted to physical signs and their interpretation. The author sets out to show the art of diagnosis by clinical methods without resort to radiographic or pathological examination, and to transcribe into book form a method of teaching which is wholly admirable when applied to the oral instruction of junior students in an out-patient department.

The basic signs upon which our recognition of the nature and intensity of disease is founded are so few in number that the fire and enthusiasm of the teacher are apt to be damped by repetition when they are transferred to print. In trying to avoid this misfortune the author has introduced descriptive terms not in current use and therefore demanding memorization of their definitions. On p. 19 inflammatory swellings are classified as transient, initial, transitional, and terminal, but in the discussion which follows a certain effort is necessary to to remember that the initial inflammatory swellings have necrosis as their chief pathological basis.

An attempt to secure emphasis by the use of idiosyncratic terminology is seen on p. 15, where transmitted pulsation becomes projectile pulsation. The use of such mistaken condensations as "craggy abscess" on p. 11 can only be deplored. The author has not entirely escaped the pitfall of mistaking the want of knowledge of the new student for a want of intelligence. In attempting to simplify the examination of swellings of the breast he is led to state on p. 225 that, as fat at body temperature is fluid, the liquid breast tissue is displaced by pressure from over a carcinoma. On p. 221 his reference to swellings "poised in the limpid fat of the breast" may be rhetorical, but is none the less misleading.

The statements that cavernous lymphangiomata "all originate deep to the deep fascia" on p. 71, and on p. 182 that they are "characterized essentially by a gross distention of the glands with lymph", arouse the suspicion that the author's avoidance of pathological discussion is not wholly founded on recognition of the greater value of physical signs.

The book is generously illustrated by diagrams of conventional simplicity, in which emphasis on a desired point is too often achieved at the expense of accuracy in the rest of the drawing. Examples of this tendency are *Fig.* 12 on p. 21, *Fig.* 17 on p. 29, *Fig.* 20 on p. 45, *Fig.* 22 on p. 60, *Fig.* 35 on p. 104, and *Fig.* 66 on p. 179.

An attractive feature is the insertion at the end of each chapter of short biographical notes on men famous in anatomy, pathology, and surgery.

Diverticula and Diverticulitis of the Intestine. Their Pathology, Diagnosis, and Treatment. By HAROLD C. EDWARDS, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon and Lecturer in Surgery to King's College Hospital, London; etc. 9 × 6 in. Pp. 335 + xii, with 223 illustrations, many in colour. 1939. Bristol: John Wright & Sons Ltd. 25s. net.

THIS book is based upon the Jacksonian Prize Essay of the Royal College of Surgeons for the year 1932, but during the seven years that have since elapsed the author has revised and added to the scope of the essay, acquired much fresh material, and given us a book that is

a complete summary of the subject. Because of his known interest in diverticula he has been able to observe an unusually large series of personal cases, but he has also investigated the clinical notes of many more under the care of his colleagues and has utilized the material provided by radiographs, post-mortem investigations, and museum specimens for information and illustration. In addition he has consulted the writings of surgeons in all countries, and presents a very comprehensive bibliography.

Mr. Edwards adopts the classification of diverticula into the congenital and the acquired as the only possible one in this difficult subject; and in the acquired group, which form the great majority, he deals with his material on a regional basis. In each group he discusses the incidence, morbid anatomy, pathogenesis, clinical aspects, diagnosis, complications, and treatment. The argument in the text is amplified by excellent diagrams and the reproduction of microscopical sections, the majority in colour. Of particular interest are the chapters on diverticula of the first part of the duodenum, which include ulcer diverticula and pulsion diverticula beside the ampulla of Vater, and those on diverticula of the large intestine which, as might be expected, take nearly half the book. The discussion on the indications for surgery, and the detailed advice upon operative methods in this last section, are particularly helpful.

This work gives the reviewer the comfortable impression that it contains all that can usefully be said about the subject with which it deals. Mr. Edwards has taken many years and much trouble to make it as complete as it can be, and he writes throughout, of the reports of others and of his own observations, with the sane and critical outlook of a mature and experienced surgeon. The illustrations are carefully chosen, well reproduced, and placed exactly where they best help to elucidate the text. Here is a masterly book on an important subject.

Diagnosis and Management of Diseases of the Biliary Tract. By R. FRANKLIN CARTER, B.S., M.D., F.A.C.S.; CARL H. GREENE, A.B., Ph.D,. M.D., F.A.C.P.; and JOHN RUSSELL TWISS, A.B., M.D., F.A.C.P., New York Post-Graduate Medical School, Columbia University. 9¼ × 6 in. Pp. 432, with 84 illustrations and 6 plates. 1939. London: Baillière, Tindall & Cox. 32s. 6d. net.

THE principal purpose of this book is, as the authors state, to present a brief treatise on present-day concepts of the basic factors in gall-bladder disease and an outline of those methods of investigation which will provide for a division of patients into those who are to be treated surgically and those who are to be treated medically. The factors upon which these recommendations are made have been derived from the follow-up findings on patients who were subjected to a division and management described in this book.

The book is divided into six parts. The first is mainly an up-to-date résumé of the physiology of the biliary tract and its nervous regulation. There is a good chapter here on the different kinds of gall-bladder associated with stasis. Considerations of the causes of gall-stone formation and jaundice and the clinical use of hepatic function tests follow usually accepted ideas; in this part of the work is included a very valuable statistical study of 926 cases of jaundice associated with disease of the liver and gall-bladder.

Part 2 deals with the diagnostic investigation of patients with disease of the biliary tract, and it is evident that the authors regard duodenal drainage as a method of great significance. They give a full description of the procedure and discuss at length the meaning of the findings. There is a good chapter on the technique of hepatic function tests and biochemical methods of investigation, and a valuable chapter on colon bacillus infection in diseases of the biliary tract.

Part 3 describes in detail medical treatment and also post-operative medical management. Several comprehensive diet sheets are set out, with the reasons for their use.

Parts 4 and 5 are concerned with surgical management and results.

These sections of the book are divided into chapters dealing with functional disorders associated with non-calculous infection, and infection accompanied by stones; in each case the operative results are given, with the follow-up findings. In the calculous cases the progressive pathology and its relation to liver injury is well described, and the reasons given for the increasing risk associated with delay in instituting surgical therapy. The technique of cholangiography and the interpretation of the cholangiograms are considered, and there

is useful information as to when drainage tubes in the gall-bladder and common duct should be removed.

Finally Part 6 is inserted as an appendix, listing the cholesterol, fat, carbohydrate, and protein-containing foods.

We regard this book as a very valuable contribution to the subjects with which it deals. It is a book for physicians and surgeons alike; for the latter the results of operative experience in general hospital service for gall-bladder disease as regards both mortality and morbidity is of special significance; and coming from men devoting special attention to this branch of surgery a close study cannot fail to be of great benefit and of unusual interest.

Patologia y Cirugia del Espinter de Oddi. By DELFOR DEL VALLE, Professor Extraordinary of Clinical Surgery, Gastroenterological Institute of Hospital Ramos Mejía. 9 × 6½ in. Pp. 275, with 55 illustrations. 1939. Buenos Aires: El Ateneo.

THE sub-heading of this monograph, "Chronic sclero-retractile choledocho-odditis, and its relations to the syndromes of cholelithiasis, primary biliary stagnation, and acute and chronic pancreatitis", well indicates its extent. The author has long insisted on the importance of persistent inflammation of the ducts, the ampulla, and the papilla, after operation for gall-stones; in fact, though he practises cholecystectomy, he still has a liking for continued drainage of the common duct, which he maintains sometimes for as long as four weeks. He has also for the past fifteen years studied the inflammatory, ulcerative, sclerotic, and retractile state of the papilla of Vater and of the ampulla, and he here figures the various conditions to be found.

He defines and distinguishes the symptomatology of obstruction due to sclerosis, suggests the possibility of similar symptoms the result of mere spasm, and differentiates the clinical indications of infection in the biliary tract arising at its termination from that originating in the gall-bladder. He is of opinion that infection frequently reaches the tract, not only via the blood-stream, but also along the duct direct from the duodenum, in the presence of sclerosis and rigidity of the terminal part.

He finds *B. pyocyaneus* and anaerobic organisms second only in frequency to *B. coli*.

He details animal experiments which throw light upon the effects which infection and obstruction to the outflow of bile have upon the liver, and records the detailed history of 24 cases.

A sound piece of work.

Operative Orthopedics. By WILLIS C. CAMPBELL, M.D., Memphis, Tenn. 9½ × 6½ in. Pp. 1154 + xix, with 845 illustrations, including 4 coloured plates. 1939. London: Henry Kimpton. 55s. net.

THIS carefully produced work is assured of a welcome. Most large works at the present time are of multiple authorship, lose coherence, often overlap, and even contain contradictions. The contrast with this unified product of one who in classical language may be described as "of riper years" is striking. The writer, a master of his subject, has produced the result of mature experience. In days when operations are described immediately after performance it is pleasant to see photographs and radiographs labelled twenty-two years after operation. While the title and main theme is operative procedures, Dr. Campbell has not restricted his descriptions in too pedantic a fashion, and makes wise use of pathological description in the text and has included a comprehensive survey of bone anatomy and physiology as his opening chapter. For such a large work the introduction is surprisingly brief, but important, as it gives a nomenclature of positions of joints and range of motion that requires frequent reference in subsequent chapters. This nomenclature is a little different from the orthodox English one and it is therefore the more important to note the differences. Dr. Campbell departs from his title to give us a valuable chapter on Apparatus. A chapter on Surgical Technique follows. The essence of the work is then entered with a beautifully clear chapter on Surgical Approaches. This will prove invaluable to the younger operators. The excellent descriptions of A. K. Henry have been given proper prominence. This chapter, one of the key chapters, could almost be larger. It is a pity that some of the illustrations proper to the chapter are found elsewhere in the book, illustrating some detailed procedure. Thus while

two illustrations demonstrate approach to the knee-joint, other approaches are depicted under the section dealing with excision of semilunar cartilages and arthrodesis, etc. However, the cross-referencing is adequate and presumably cost prohibits duplicating. In some parts of the book the plethora of description is almost overwhelming; thus five approaches to the os calcis are given, eight operations for recurrent dislocation of the patella, and so on. The less experienced surgeon, confronted with such a choice, may be well advised to select the procedure which has the name Campbell in brackets after the title. He can then feel assured that the method has been frequently employed and has stood the criticism of the years. A valuable chapter on Arthrodesis follows three chapters devoted to Acute and Low-grade Affections of Joints. Dr. Campbell prefaces many of these central chapters with a helpful general discussion of the problems on technique involved. This is well shown in the introduction to the succeeding chapter of Arthroplasty. It is almost to be wished that a similar discussion on selection of cases suitable for operation had been included, though this would make a large book perhaps completely unwieldy.

Two hundred pages in the centre of the book are devoted to operative measures for fractures and dislocations. A perusal of these pages almost tempts one to forget the closed methods of treatment. Such oblivion would not be Dr. Campbell's responsibility, as throughout the book, and even as the opening sentences of his introduction, he is careful to point out that the majority of orthopædic conditions may often be relieved without operation. At a time when it is almost anathema to operate on fractures, it is refreshing to read what can be done with the difficult case or unsatisfactory result. Cases which one might call untreatable are often amenable to some relief, sometimes not inconsiderable, by an operative procedure.

The section on fractures is followed by chapters on Acute and Low-grade Affections of Bones, Tumours of Bones and Soft Tissues, Affections of Muscles, Tendons, and Tendon sheaths. The reader is then treated to a delightful section on plastic surgery in the chapter on Affections of the Skin, Fascia, Bursæ, etc. Affections of the Nervous System, 180 pages, Static or Postural Affections, and Congenital Anomalies complete the volume. A description of amputations has not been included.

After the above general appreciation which is meant to be generous and acclamative, the following detailed criticism seems almost carping. A careful reading of the book detected only one misprint: "base" for "head" of metatarsal on p. 859.

In the section on apparatus there is no description of the very useful Trumble walking-calliper, and while most elaborate fracture tables are shown, there is no description of the very simple 'two-table' method (Watson Jones) for the application of plaster jackets. The photograph of the plaster jacket in *Fig.* 52 seems wrong. The Americanism 'infectious' for 'infective' crops up in an irritating manner. The references to Böhler's book are to the out-of-date edition. The section on paraplegia, p. 272, makes no use of Seddon and Butler's valuable work, though the papers are indexed.

In the description of tendon lengthening of the Achilles tendon (p. 330), no mention is made of the very real risk of over-lengthening; perhaps this is reasonable, as on p. 967 the author states he has never seen calcaneus result. The description of the McMurray type of osteotomy is rather poor for such a popular operation—no diagram or figure included. For repair of the anterior cruciate ligament, six drawings of Campbell's operation are included, but not a single sketch of the classical operation of Hey Groves. The single page devoted to anæsthesia could well be deleted as it is of no constructive value.

The allusions to chemotherapy are out of date, but that is hardly a matter for comment in such a changing branch of therapeutics. With regard to the vexed question of the time to operate in acute hæmatogenous osteomyelitis, Dr. Campbell is among the 'delayed school' advocates, but while admittedly he is in good company he does not present the case very fairly. In the section on tumours he discusses the occasional need for interscapulo-thoracic amputation, but he does not mention or include in the references interinnomino-iliac amputation, successfully performed by Gordon-Taylor and others.

The section on Lambrinudi's operation is rather inadequate for such a clever and important operation. It is reasonable that Dr. Campbell should prefer his own ingenious bone-stop operation, but Lambrinudi's procedure is not less ingenious and moreover calls for the most careful execution.

The above are minor comments on a book to be welcomed as a major work of a high order.

Infections of the Hand. By L. R. FIFIELD, F.R.C.S. Eng. Second edition, by PATRICK CLARKSON, F.R.C.S. Eng., Surgical Tutor, Guy's Hospital; Demonstrator of Anatomy and Operative Surgery, Guy's Hospital Medical School. 7¼ × 4⅞ in. Pp. 168 + xii, with 57 illustrations, including 8 plates (2 coloured). 1939. London: H. K. Lewis & Co. Ltd. 9s. net.

THE subject of hand infections is so large and so important that it must be described in a detail for which the space allotted in surgical text-books is inadequate. A monograph is necessary, and for many years Fifield's book has had the reputation of containing all the essential practical information in compact form. A fresh edition has been called for by the continued demand, and because of the necessity to modify the surgical recommendations in the light of ten years' experience and to include new methods that have appeared, notably the use of chemotherapy.

Mr. Clarkson, who has had charge of the minor surgical department of a large teaching hospital, is particularly well suited for this task. He has brought Fifield's original contributions up to date, and has added chapters on prophylaxis, including the treatment of wounds and punctures of the hands; on acute diffuse infections; infections of the dorsum; infections with unusual organisms such as anthrax; on treatment in general; and on prognosis. The anatomical parts of the book, which are based on Kanavel's work, need no discussion: Fifield's chapters were good, and are still better by revision. Mr. Clarkson's own contributions are admirably lucid and practical. The two last chapters—that on treatment, which includes re-education and management of complications, and that on prognosis—are particularly clear and to the point.

This edition of Fifield's book will enhance the reputation of the former, whose familiar appearance it reproduces. Though slightly thinner, it manages by finer paper and different type to get a good deal more between its covers. The illustrations are well chosen and helpful, though some of the line diagrams could be improved by greater contrast and a less conventional indication of the subcutaneous layer.

Infections of the Hand can be recommended as a clear and comprehensive treatise on the practical aspects of an important branch of surgery with which every student and practitioner must be conversant.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Diseases of the Foot. By EMIL D. W. HAUSER, M.S., M.D., Assistant Professor of Bone and Joint Surgery, Northwestern University Medical School; Attending Orthopædic Surgeon, Passavant Memorial Hospital, Chicago. With a Foreword by SUMNER L. KOCH, M.D. 9⅛ × 6 in. Pp. 472 + xv, with 173 illustrations, some in colour. 1939. Philadelphia and London: W. B. Saunders Co. 27s. 6d. net.

Minor Surgery. By R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon, Royal Northern and Metropolitan Hospitals; etc. 7¼ × 4⅞ in. Pp. 379 + viii, with 155 illustrations. 1940. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.

Minor Surgery and the Treatment of Fractures (Heath: Pollard: Davies). By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. With a chapter on "The Administration of Anæsthetics" by H. N. WEBBER, B.Chir., D.A., Anæsthetist to University College Hospital. Twenty-second edition. 7¼ × 4⅞ in. Pp. 472 + viii, with 283 illustrations. 1940. London: J. & A. Churchill. 12s. 6d. net.

The Pineal Organ. By REGINALD J. GLADSTONE, M.D., F.R.C.S., F.R.S.E., D.P.H., Formerly Reader in Embryology and Lecturer on Anatomy at King's College, University of London, and Lecturer on Embryology at the Medical College, Middlesex Hospital, London; and CECIL P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E., F.Z.S., F.A.C.S., F.R.A.C.S., Fellow of King's College, London, and Lecturer in Applied Anatomy at King's College, University of London; Senior Surgeon, King's College Hospital and the West End Hospital for Nervous Diseases; etc. 10 × 6½ in. Pp. 528 + xvi, with 324 illustrations. 1940. London: Baillière, Tindall & Cox. 42s. net.

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PRINTED IN GREAT BRITAIN BY JOHN WRIGHT AND SONS LTD., BRISTOL

VOL. XXVII QUARTERLY No. 108

Medical Library JUN 10 1940

The British Journal of Surgery

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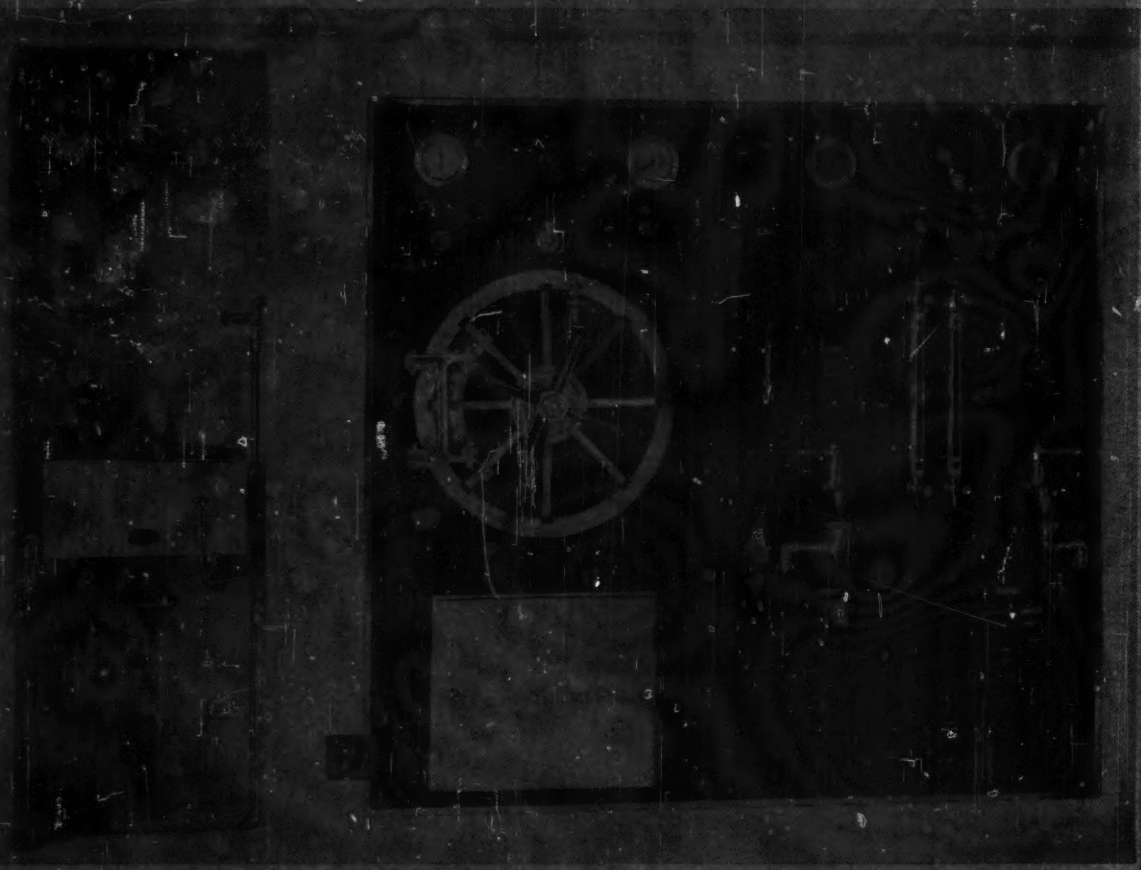

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